4410609 NO4/10

STIC-ILL

440501

From: Prouty, Rebecca
Sent: Thursday, April 10, 2003 2:06 PM
To: STIC-ILL
Subject: ILL Request

Art Unit 1652
10A13, 308-4000
Mailbox: 10D01
Serial Number: 09/829,545

Please provide the following reference(s):

L118 ANSWER 4 OF 12 MEDLINE DUPLICATE 2
TI Polymorphisms of N-acetyltransferases, glutathione S-transferases, microsomal epoxide hydrolase and sulfotransferases: influence on cancer susceptibility.
SO RECENT RESULTS IN CANCER RESEARCH, (1998) 154 47-85. Ref: 178
Journal code: 0044671. ISSN: 0080-0015.
AU Hengstler J G; Arand M; Herrero M E; Oesch F
AN 1999151055 MEDLINE

1052266

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$ * * * * * * * * * * * * * STN Columbus * * * * * * * * * * * * * * * *

FILE 'HOME' ENTERED AT 09:54:44 ON 10 APR 2003

=> fil .bec,canc
COST IN U.S. DOLLARS                                  SINCE FILE     TOTAL
                                                           ENTRY   SESSION
FULL ESTIMATED COST                                         0.21      0.21

FILES 'MEDLINE, SCISEARCH, LIFESCI, BIOTECHDS, BIOSIS, EMBASE, HCAPLUS, NTIS,
       ESBIOBASE, BIOTECHNO, WPIDS, CANCERLIT' ENTERED AT 09:55:08 ON 10 APR 2003
ALL COPYRIGHTS AND RESTRICTIONS APPLY.  SEE HELP USAGETERMS FOR DETAILS.

12 FILES IN THE FILE LIST

=> s sulfotransferase#
FILE 'MEDLINE'
L1          1967 SULFOTRANSFERASE#

FILE 'SCISEARCH'
L2          2394 SULFOTRANSFERASE#

FILE 'LIFESCI'
L3           596 SULFOTRANSFERASE#

FILE 'BIOTECHDS'
L4            99 SULFOTRANSFERASE#

FILE 'BIOSIS'
L5          2652 SULFOTRANSFERASE#

FILE 'EMBASE'
L6          2142 SULFOTRANSFERASE#

FILE 'HCAPLUS'
L7          2954 SULFOTRANSFERASE#

FILE 'NTIS'
L8            15 SULFOTRANSFERASE#

FILE 'ESBIOBASE'
L9           786 SULFOTRANSFERASE#

FILE 'BIOTECHNO'
L10          945 SULFOTRANSFERASE#

FILE 'WPIDS'
L11           79 SULFOTRANSFERASE#

FILE 'CANCERLIT'
L12          471 SULFOTRANSFERASE#

TOTAL FOR ALL FILES
L13        15100 SULFOTRANSFERASE#

=> s l13(5a)phenol?
FILE 'MEDLINE'
           30780 PHENOL?
L14          268 L1 (5A)PHENOL?

FILE 'SCISEARCH'
           59114 PHENOL?
L15          545 L2 (5A)PHENOL?
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FILE 'LIFESCI'
          16034 PHENOL?
L16          96 L3  (5A)PHENOL?

FILE 'BIOTECHDS'
          10933 PHENOL?
L17           5 L4  (5A)PHENOL?

FILE 'BIOSIS'
          63668 PHENOL?
L18         405 L5  (5A)PHENOL?

FILE 'EMBASE'
          28609 PHENOL?
L19         254 L6  (5A)PHENOL?

FILE 'HCAPLUS'
         353836 PHENOL?
L20         516 L7  (5A)PHENOL?

FILE 'NTIS'
           6569 PHENOL?
L21           0 L8  (5A)PHENOL?

FILE 'ESBIOBASE'
          21653 PHENOL?
L22         120 L9  (5A)PHENOL?

FILE 'BIOTECHNO'
           9384 PHENOL?
L23         112 L10(5A)PHENOL?

FILE 'WPIDS'
         109748 PHENOL?
L24           4 L11(5A)PHENOL?

FILE 'CANCERLIT'
           3833 PHENOL?
L25          41 L12(5A)PHENOL?

TOTAL FOR ALL FILES
L26        2366 L13(5A)  PHENOL?

=> s l13 and pst
FILE 'MEDLINE'
           1643 PST
L27         157 L1 AND PST

FILE 'SCISEARCH'
           1888 PST
L28         158 L2 AND PST

FILE 'LIFESCI'
            654 PST
L29          60 L3 AND PST

FILE 'BIOTECHDS'
            142 PST
L30           1 L4 AND PST

FILE 'BIOSIS'
           2577 PST
L31         187 L5 AND PST
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FILE 'EMBASE'
          1349 PST
L32        202 L6 AND PST

FILE 'HCAPLUS'
          2200 PST
L33        183 L7 AND PST

FILE 'NTIS'
           116 PST
L34          0 L8 AND PST

FILE 'ESBIOBASE'
           453 PST
L35         65 L9 AND PST

FILE 'BIOTECHNO'
           642 PST
L36         84 L10 AND PST

FILE 'WPIDS'
           407 PST
L37          0 L11 AND PST

FILE 'CANCERLIT'
           266 PST
L38         28 L12 AND PST

TOTAL FOR ALL FILES
L39       1125 L13 AND PST

=> s 126 or 139
FILE 'MEDLINE'
L40        309 L14 OR L27

FILE 'SCISEARCH'
L41        561 L15 OR L28

FILE 'LIFESCI'
L42        111 L16 OR L29

FILE 'BIOTECHDS'
L43          6 L17 OR L30

FILE 'BIOSIS'
L44        440 L18 OR L31

FILE 'EMBASE'
L45        340 L19 OR L32

FILE 'HCAPLUS'
L46        544 L20 OR L33

FILE 'NTIS'
L47          0 L21 OR L34

FILE 'ESBIOBASE'
L48        139 L22 OR L35

FILE 'BIOTECHNO'
L49        144 L23 OR L36

FILE 'WPIDS'
L50          4 L24 OR L37
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FILE 'CANCERLIT'
L51          52 L25 OR L38

TOTAL FOR ALL FILES
L52        2650 L26 OR L39

=> s hormone(3a)depend?
FILE 'MEDLINE'
         213298 HORMONE
         842930 DEPEND?
L53        7312 HORMONE(3A)DEPEND?

FILE 'SCISEARCH'
         191023 HORMONE
        1152609 DEPEND?
L54        3447 HORMONE(3A)DEPEND?

FILE 'LIFESCI'
          47329 HORMONE
         265495 DEPEND?
L55        1195 HORMONE(3A)DEPEND?

FILE 'BIOTECHDS'
           7327 HORMONE
          18376 DEPEND?
L56          61 HORMONE(3A)DEPEND?

FILE 'BIOSIS'
         417137 HORMONE
         919113 DEPEND?
L57        5558 HORMONE(3A)DEPEND?

FILE 'EMBASE'
         257543 HORMONE
         796102 DEPEND?
L58        5066 HORMONE(3A)DEPEND?

FILE 'HCAPLUS'
         239881 HORMONE
        1988234 DEPEND?
L59        5310 HORMONE(3A)DEPEND?

FILE 'NTIS'
           1912 HORMONE
         133348 DEPEND?
L60          63 HORMONE(3A)DEPEND?

FILE 'ESBIOBASE'
          53120 HORMONE
         292504 DEPEND?
L61        1400 HORMONE(3A)DEPEND?

FILE 'BIOTECHNO'
          71939 HORMONE
         232777 DEPEND?
L62        1849 HORMONE(3A)DEPEND?

FILE 'WPIDS'
          12836 HORMONE
         270843 DEPEND?
L63         509 HORMONE(3A)DEPEND?

FILE 'CANCERLIT'
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          50714 HORMONE
         180961 DEPEND?
L64        5215 HORMONE(3A)DEPEND?

TOTAL FOR ALL FILES
L65       36985 HORMONE(3A) DEPEND?

=> s l52 and l65
FILE 'MEDLINE'
L66           0 L40 AND L53

FILE 'SCISEARCH'
L67           1 L41 AND L54

FILE 'LIFESCI'
L68           0 L42 AND L55

FILE 'BIOTECHDS'
L69           0 L43 AND L56

FILE 'BIOSIS'
L70           0 L44 AND L57

FILE 'EMBASE'
L71           0 L45 AND L58

FILE 'HCAPLUS'
L72           1 L46 AND L59

FILE 'NTIS'
L73           0 L47 AND L60

FILE 'ESBIOBASE'
L74           0 L48 AND L61

FILE 'BIOTECHNO'
L75           0 L49 AND L62

FILE 'WPIDS'
L76           0 L50 AND L63

FILE 'CANCERLIT'
L77           0 L51 AND L64

TOTAL FOR ALL FILES
L78           2 L52 AND L65

=> s l52 and risk
FILE 'MEDLINE'
         554461 RISK
L79           7 L40 AND RISK

FILE 'SCISEARCH'
         372816 RISK
L80          18 L41 AND RISK

FILE 'LIFESCI'
          52096 RISK
L81           4 L42 AND RISK

FILE 'BIOTECHDS'
           2383 RISK
L82           0 L43 AND RISK
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FILE 'BIOSIS'
        331098 RISK
L83          9 L44 AND RISK

FILE 'EMBASE'
        485074 RISK
L84          8 L45 AND RISK

FILE 'HCAPLUS'
        101333 RISK
L85         19 L46 AND RISK

FILE 'NTIS'
         39545 RISK
L86          0 L47 AND RISK

FILE 'ESBIOBASE'
        105005 RISK
L87          7 L48 AND RISK

FILE 'BIOTECHNO'
         44575 RISK
L88          3 L49 AND RISK

FILE 'WPIDS'
         63874 RISK
L89          0 L50 AND RISK

FILE 'CANCERLIT'
        122495 RISK
L90          3 L51 AND RISK

TOTAL FOR ALL FILES
L91         78 L52 AND RISK

=> s 152 and cancer
FILE 'MEDLINE'
        405957 CANCER
L92          9 L40 AND CANCER

FILE 'SCISEARCH'
        416574 CANCER
L93         40 L41 AND CANCER

FILE 'LIFESCI'
         43287 CANCER
L94          3 L42 AND CANCER

FILE 'BIOTECHDS'
         20197 CANCER
L95          1 L43 AND CANCER

FILE 'BIOSIS'
        424817 CANCER
L96         16 L44 AND CANCER

FILE 'EMBASE'
        662305 CANCER
L97         13 L45 AND CANCER

FILE 'HCAPLUS'
        183865 CANCER
L98         27 L46 AND CANCER
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FILE 'NTIS'
        13892 CANCER
L99         0 L47 AND CANCER

FILE 'ESBIOBASE'
       230130 CANCER
L100       14 L48 AND CANCER

FILE 'BIOTECHNO'
       109592 CANCER
L101        7 L49 AND CANCER

FILE 'WPIDS'
        38222 CANCER
L102        1 L50 AND CANCER

FILE 'CANCERLIT'
       394414 CANCER
L103        6 L51 AND CANCER

TOTAL FOR ALL FILES
L104      137 L52 AND CANCER

=> s l52 and tumor?
FILE 'MEDLINE'
       617642 TUMOR?
L105       18 L40 AND TUMOR?

FILE 'SCISEARCH'
       442978 TUMOR?
L106        7 L41 AND TUMOR?

FILE 'LIFESCI'
        83852 TUMOR?
L107        1 L42 AND TUMOR?

FILE 'BIOTECHDS'
        27345 TUMOR?
L108        1 L43 AND TUMOR?

FILE 'BIOSIS'
       674967 TUMOR?
L109       30 L44 AND TUMOR?

FILE 'EMBASE'
       566893 TUMOR?
L110        6 L45 AND TUMOR?

FILE 'HCAPLUS'
       320911 TUMOR?
L111       19 L46 AND TUMOR?

FILE 'NTIS'
         7146 TUMOR?
L112        0 L47 AND TUMOR?

FILE 'ESBIOBASE'
       116937 TUMOR?
L113        2 L48 AND TUMOR?

FILE 'BIOTECHNO'
       137139 TUMOR?
L114        3 L49 AND TUMOR?
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FILE 'WPIDS'
         17974 TUMOR?
L115         1 L50 AND TUMOR?

FILE 'CANCERLIT'
        629285 TUMOR?
L116        18 L51 AND TUMOR?

TOTAL FOR ALL FILES
L117       106 L52 AND TUMOR?

=> s l78 or l91 or l104 or l117
FILE 'MEDLINE'
L118        26 L66 OR L79 OR L92 OR L105

FILE 'SCISEARCH'
L119        50 L67 OR L80 OR L93 OR L106

FILE 'LIFESCI'
L120         6 L68 OR L81 OR L94 OR L107

FILE 'BIOTECHDS'
L121         1 L69 OR L82 OR L95 OR L108

FILE 'BIOSIS'
L122        37 L70 OR L83 OR L96 OR L109

FILE 'EMBASE'
L123        21 L71 OR L84 OR L97 OR L110

FILE 'HCAPLUS'
L124        46 L72 OR L85 OR L98 OR L111

FILE 'NTIS'
L125         0 L73 OR L86 OR L99 OR L112

FILE 'ESBIOBASE'
L126        18 L74 OR L87 OR L100 OR L113

FILE 'BIOTECHNO'
L127         9 L75 OR L88 OR L101 OR L114

FILE 'WPIDS'
L128         1 L76 OR L89 OR L102 OR L115

FILE 'CANCERLIT'
L129        21 L77 OR L90 OR L103 OR L116

TOTAL FOR ALL FILES
L130       236 L78 OR L91 OR L104 OR L117

=> dup rem l130
PROCESSING COMPLETED FOR L130
L131       109 DUP REM L130 (127 DUPLICATES REMOVED)

=> d tot

L131 ANSWER 1 OF 109  HCAPLUS  COPYRIGHT 2003 ACS
TI   Gene expression profile biomarkers and therapeutic targets for brain aging
     and age-related cognitive impairment in rats
SO   PCT Int. Appl., 84 pp.
     CODEN: PIXXD2
IN   Landfield, Philip W.; Blalock, Eric M.; Chen, Kuey-Chu; Foster, Thomas C.
AN   2003:242437  HCAPLUS
```

```
     PATENT NO.        KIND  DATE          APPLICATION NO.  DATE
     ---------------   ----  --------      ---------------  --------
PI   WO 2003025122      A2   20030327      WO 2002-US25607  20020813
         W:  AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN,
             CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH,
             GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR,
             LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH,
             PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ,
             UA, UG, US, UZ, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU,
             TJ, TM
         RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, BG,
             CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL,
             PT, SE, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR,
             NE, SN, TD, TG
```

L131 ANSWER 2 OF 109 HCAPLUS COPYRIGHT 2003 ACS
TI Compositions and methods for inhibiting human immunodeficiency virus infection by down-regulating human cellular genes, and inhibitor identification methods
SO PCT Int. Appl., 44 pp.
CODEN: PIXXD2
IN Holzmayer, Tanya A.; Dunn, Stephen J.
AN 2003:22845 HCAPLUS
DN 138:83340

```
     PATENT NO.        KIND  DATE          APPLICATION NO.  DATE
     ---------------   ----  --------      ---------------  --------
PI   WO 2003002528      A2   20030109      WO 2002-US20964  20020701
         W:  AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN,
             CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH,
             GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR,
             LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH,
             PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ,
             UA, UG, US, UZ, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU,
             TJ, TM
         RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, BG,
             CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL,
             PT, SE, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR,
             NE, SN, TD, TG
```

L131 ANSWER 3 OF 109 SCISEARCH COPYRIGHT 2003 ISI (R)
TI Structure of a human carcinogen-converting enzyme, SULT1A1 - Structural and kinetic implications of substrate inhibition
SO JOURNAL OF BIOLOGICAL CHEMISTRY, (28 FEB 2003) Vol. 278, No. 9, pp. 7655-7662.
Publisher: AMER SOC BIOCHEMISTRY MOLECULAR BIOLOGY INC, 9650 ROCKVILLE PIKE, BETHESDA, MD 20814-3996 USA.
ISSN: 0021-9258.
AU Gamage N U; Duggleby R G; Barnett A C; Tresillian M; Latham C F; Liyou N E; McManus M E; Martin J L (Reprint)
AN 2003:198648 SCISEARCH

L131 ANSWER 4 OF 109 HCAPLUS COPYRIGHT 2003 ACS
TI SULT1A1 polymorphism and esophageal **cancer** in males
SO International Journal of Cancer (2003), 103(1), 101-104
CODEN: IJCNAW; ISSN: 0020-7136
AU Wu, Ming-Tsang; Wang, Yi-Ting; Ho, Chi-Kung; Wu, Deng-Chyang; Lee, Yung-Chie; Hsu, Hon-Ki; Kao, Ein-Long; Lee, Jang-Ming
AN 2002:939248 HCAPLUS
DN 138:151235

L131 ANSWER 5 OF 109 WPIDS (C) 2003 THOMSON DERWENT
TI Use of a steroid derivative of 5-androstene, 5-pregnenolone or corresponding saturated derivatives in the treatment and/or prevention of a benign and/or malignant **tumor**.

PI WO 2002072003 A2 20020919 (200276)* EN 54p A61K000-00
RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ NL OA PT SD SE SL SZ TR TZ UG ZM ZW
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CO CR CU CZ DE DK DM DZ EC EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ OM PH PL PT RO RU SD SE SG SI SK SL TJ TM TN TR TT TZ UA UG US UZ VN YU ZA ZM ZW
IN HAGSTROEM, T

L131 ANSWER 6 OF 109 HCAPLUS COPYRIGHT 2003 ACS
TI Reverse geometrical selectivity in glucuronidation and sulfation of cis- and trans-4-hydroxytamoxifens by human liver UDP-glucuronosyltransferases and sulfotransferases
SO Biochemical Pharmacology (2002), 63(10), 1817-1830
CODEN: BCPCA6; ISSN: 0006-2952
AU Nishiyama, Takahito; Ogura, Kenichiro; Nakano, Hiroaki; Ohnuma, Tomokazu; Kaku, Teppei; Hiratsuka, Akira; Muro, Kei; Watabe, Tadashi
AN 2002:404159 HCAPLUS
DN 138:117212

L131 ANSWER 7 OF 109 SCISEARCH COPYRIGHT 2003 ISI (R)
TI Association between sulfotransferase 1A1 genotype and survival of breast **cancer** patients receiving tamoxifen therapy
SO JOURNAL OF THE NATIONAL CANCER INSTITUTE, (6 NOV 2002) Vol. 94, No. 21, pp. 1635-1640.
Publisher: OXFORD UNIV PRESS INC, JOURNALS DEPT, 2001 EVANS RD, CARY, NC 27513 USA.
ISSN: 0027-8874.
AU Nowell S (Reprint); Sweeney C; Winters M; Stone A; Lang N P; Hutchins L F; Kadlubar F F; Ambrosone C B
AN 2002:911183 SCISEARCH

L131 ANSWER 8 OF 109 HCAPLUS COPYRIGHT 2003 ACS
TI Identification of genes regulated by dexamethasone in multiple myeloma cells using oligonucleotide arrays
SO Oncogene (2002), 21(9), 1346-1358
CODEN: ONCNES; ISSN: 0950-9232
AU Chauhan, Dharminder; Auclair, Daniel; Robinson, Elisabeth K.; Hideshima, Teru; Li, Guilan; Podar, Klaus; Gupta, Deepak; Richardson, Paul; Schlossman, Robert L.; Krett, Nancy; Chen, Lan Bo; Munshi, Nikhil C.; Anderson, Kenneth C.
AN 2002:214668 HCAPLUS
DN 137:150359

L131 ANSWER 9 OF 109 SCISEARCH COPYRIGHT 2003 ISI (R)
TI Glucuronidation and sulfation of the tea flavonoid (-)-epicatechin by the human and rat enzymes
SO DRUG METABOLISM AND DISPOSITION, (AUG 2002) Vol. 30, No. 8, pp. 897-903.
Publisher: AMER SOC PHARMACOLOGY EXPERIMENTAL THERAPEUTICS, 9650 ROCKVILLE PIKE, BETHESDA, MD 20814-3998 USA.
ISSN: 0090-9556.
AU Vaidyanathan J B; Walle T (Reprint)
AN 2002:610531 SCISEARCH

L131 ANSWER 10 OF 109 HCAPLUS COPYRIGHT 2003 ACS
TI Transcript profiling of enzymes involved in detoxification of xenobiotics and reactive oxygen in human normal and simian virus 40 T antigen-immortalized oral keratinocytes
SO International Journal of Cancer (2002), 99(6), 776-782
CODEN: IJCNAW; ISSN: 0020-7136
AU Vondracek, Martin; Weaver, David A.; Sarang, Zsolt; Hedberg, Jesper J.; Willey, James C.; Warngard, Lars; Grafstrom, Roland C.
AN 2002:442010 HCAPLUS

DN 138:70724

L131 ANSWER 11 OF 109 SCISEARCH COPYRIGHT 2003 ISI (R)
TI Association of the SULT1A1 R213H polymorphism with colorectal **cancer**
SO CLINICAL AND EXPERIMENTAL PHARMACOLOGY AND PHYSIOLOGY, (SEP 2002) Vol. 29, No. 9, pp. 754-758.
Publisher: BLACKWELL PUBLISHING ASIA, 54 UNIVERSITY ST, P O BOX 378, CARLTON, VICTORIA 3053, AUSTRALIA.
ISSN: 0305-1870.
AU Wong C F (Reprint); Liyou N; Leggett B; Young J; Johnson A; McManus M E
AN 2002:666203 SCISEARCH

L131 ANSWER 12 OF 109 SCISEARCH COPYRIGHT 2003 ISI (R)DUPLICATE 1
TI Sulfotransferase 1A2]2 is a **risk** factor for early-onset breast **cancer**
SO INTERNATIONAL JOURNAL OF MOLECULAR MEDICINE, (NOV 2002) Vol. 10, No. 5, pp. 609-612.
Publisher: PROFESSOR D A SPANDIDOS, 1, S MERKOURI ST, EDITORIAL OFFICE,, ATHENS 116 35, GREECE.
ISSN: 1107-3756.
AU Hou M F; Chen S T; Chen J C; Yeh K T; Lee T P; Chen C M; Shih M C; Lin C C; Chang J G (Reprint)
AN 2002:857108 SCISEARCH

L131 ANSWER 13 OF 109 MEDLINE DUPLICATE 2
TI Sulfation of bisphenol A abolished its estrogenicity based on proliferation and gene expression in human breast **cancer** MCF-7 cells.
SO TOXICOLOGY IN VITRO, (2002 Oct) 16 (5) 549-56.
Journal code: 8712158. ISSN: 0887-2333.
AU Shimizu M; Ohta K; Matsumoto Y; Fukuoka M; Ohno Y; Ozawa S
AN 2002633107 MEDLINE

L131 ANSWER 14 OF 109 MEDLINE DUPLICATE 3
TI Association of genotypes of carcinogen-activating enzymes, **phenol sulfotransferase** SULT1A1 (ST1A3) and arylamine N-acetyltransferase NAT2, with urothelial **cancer** in a Japanese population.
SO INTERNATIONAL JOURNAL OF CANCER, (2002 Dec 1) 102 (4) 418-21.
Journal code: 0042124. ISSN: 0020-7136.
AU Ozawa Shogo; Katoh Takahiko; Inatomi Hisato; Imai Hirohisa; Kuroda Yoshiki; Ichiba Masayoshi; Ohno Yasuo
AN 2002642601 MEDLINE

L131 ANSWER 15 OF 109 SCISEARCH COPYRIGHT 2003 ISI (R)DUPLICATE 4
TI Catecholestrogen sulfation: Possible role in carcinogenesis
SO BIOCHEMICAL AND BIOPHYSICAL RESEARCH COMMUNICATIONS, (29 MAR 2002) Vol. 292, No. 2, pp. 402-408.
Publisher: ACADEMIC PRESS INC, 525 B ST, STE 1900, SAN DIEGO, CA 92101-4495 USA.
ISSN: 0006-291X.
AU Adjei A A; Weinshilboum R M (Reprint)
AN 2002:307430 SCISEARCH

L131 ANSWER 16 OF 109 HCAPLUS COPYRIGHT 2003 ACS
TI Fish oil feeding alters liver gene expressions to defend against PPAR.alpha. activation and ROS production
SO American Journal of Physiology (2002), 282(2, Pt. 1), G338-G348
CODEN: AJPHAP; ISSN: 0002-9513
AU Takahashi, Mayumi; Tsuboyama-Kasaoka, Nobuyo; Nakatani, Teruyo; Ishii, Masami; Tsutsumi, Shuichi; Aburatani, Hiroyuki; Ezaki, Osamu
AN 2002:115193 HCAPLUS
DN 136:309178

L131 ANSWER 17 OF 109   MEDLINE   DUPLICATE 5
TI 4-Hydroxytamoxifen sulfation metabolism.
SO JOURNAL OF BIOCHEMICAL AND MOLECULAR TOXICOLOGY, (2002) 16 (6) 279-85.
Journal code: 9717231. ISSN: 1095-6670.
AU Chen Guangping; Yin Shuhua; Maiti Smarajit; Shao Xiuping
AN 2002718477   IN-PROCESS

L131 ANSWER 18 OF 109   MEDLINE   DUPLICATE 6
TI Differential xenoestrogen-sulfating activities of the human cytosolic **sulfotransferases**: molecular cloning, expression, and purification of human SULT2B1a and SULT2B1b **sulfotransferases**.
SO BIOCHIMICA ET BIOPHYSICA ACTA, (2002 Nov 14) 1573 (2) 165-70.
Journal code: 0217513. ISSN: 0006-3002.
AU Pai T Govind; Sugahara Takuya; Suiko Masahito; Sakakibara Yoichi; Xu Faye; Liu Ming-Cheh
AN 2002639685   MEDLINE

L131 ANSWER 19 OF 109 SCISEARCH COPYRIGHT 2003 ISI (R)
TI Sulfotransferase (SULT) 1A1 polymorphism as a predisposition factor for lung **cancer**: a case-control analysis
SO LUNG CANCER, (FEB 2002) Vol. 35, No. 2, pp. 137-142.
Publisher: ELSEVIER SCI IRELAND LTD, CUSTOMER RELATIONS MANAGER, BAY 15, SHANNON INDUSTRIAL ESTATE CO, CLARE, IRELAND.
ISSN: 0169-5002.
AU Wang Y F; Spitz M R; Tsou A M H; Zhang K R; Makan N; Wu X F (Reprint)
AN 2002:206680 SCISEARCH

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L131 ANSWER 21 OF 109 HCAPLUS COPYRIGHT 2003 ACS
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AN 2001:338762 HCAPLUS
DN 134:362292

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	WO 2001032928	A3	20020725		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

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L131 ANSWER 23 OF 109 HCAPLUS COPYRIGHT 2003 ACS
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L131 ANSWER 24 OF 109 HCAPLUS COPYRIGHT 2003 ACS
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CODEN: AEMBAP; ISSN: 0065-2598
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DN    135:135422

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SO    PCT Int. Appl., 43 pp.
      CODEN: PIXXD2
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DN    132:277187
      PATENT NO.         KIND  DATE          APPLICATION NO.  DATE
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PI    WO 2000020605       A1   20000413      WO 1999-US23144  19991006
          W:  AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ,
              DE, DK, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS,
              JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK,
              MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ,
              TM, TR, TT, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ,
              MD, RU, TJ, TM
          RW: GH, GM, KE, LS, MW, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE,
              DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF,
              CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG
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L131 ANSWER 37 OF 109 BIOTECHDS COPYRIGHT 2003 THOMSON DERWENT AND ISI
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L131 ANSWER 41 OF 109 HCAPLUS COPYRIGHT 2003 ACS
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L131 ANSWER 45 OF 109 LIFESCI COPYRIGHT 2003 CSA DUPLICATE 15
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L131 ANSWER 57 OF 109 HCAPLUS COPYRIGHT 2003 ACS
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L131 ANSWER 109 OF 109 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
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=> d ab 3,7,11,12,14,19,24,26,33,37,45,48,65,66,70,79,97

L131 ANSWER 3 OF 109 SCISEARCH COPYRIGHT 2003 ISI (R)
AB Sulfonation catalyzed by sulfotransferase enzymes plays an important role in chemical defense mechanisms against various xenobiotics but also bioactivates carcinogens. A major human sulfotransferase, SULT1A1, metabolizes and/or bioactivates many endogenous compounds and is implicated in a range of cancers because of its ability to modify diverse promutagen and procarcinogen xenobiotics. The crystal structure of human SULT1A1 reported here is the first sulfotransferase structure complexed with a xenobiotic substrate. An unexpected finding is that the enzyme accommodates not one but two molecules of the xenobiotic model substrate p-nitrophenol in the active site. This result is supported by kinetic data for SULT1A1 that show substrate inhibition for this small xenobiotic. The extended active site of SULT1A1 is consistent with binding of diiodothyronine but cannot easily accommodate beta-estradiol, although both are known substrates. This observation, together with evidence for a disorder-order transition in SULT1A1, suggests that the active site is flexible and can adapt its architecture to accept diverse hydrophobic substrates with varying sizes, shapes and flexibility. Thus the crystal structure of SULT1A1 provides the molecular basis for substrate inhibition and reveals the first clues as to how the enzyme sulfonates a wide variety of lipophilic compounds.

L131 ANSWER 7 OF 109 SCISEARCH COPYRIGHT 2003 ISI (R)
AB Background: Human sulfotransferase 1A1 (SULT1A1) catalyzes the sulfation of a variety of phenolic and estrogenic compounds, including 4-hydroxytamoxifen (4-OH TAM), the active metabolite of tamoxifen. A functional polymorphism in exon 7 of the SULT1A1 gene (SULT1A1*2) has been described that generates an enzyme that has approximately twofold lower activity and is less thermostable than that of the common allele SULT1A1*1. We investigated the hypothesis that that high sulfation activity would increase the elimination of 4-OH TAM by examining whether the presence of this polymorphism affects the efficacy of tamoxifen therapy. Methods: We examined the relationship between the SULT1A1*2 allele and survival in a cohort of 337 women with breast **cancer** who received tamoxifen (n = 160) or who did not (n = 177). SULT1A1 genotype was determined by restriction fragment polymorphism analysis. Patient survival was evaluated according to SULT1A1 genotype using Kaplan-Meier survival functions. Hazard ratios (HRs) were calculated from adjusted Cox proportional hazards modeling. All statistical tests were two-sided. Results: Among tamoxifen-treated patients, those who were

homozygous for the SULT1A1*2 low-activity allele had approximately three times the **risk** of death (HR = 2.9, 95% confidence interval [CI] = 1.1 to 7.6) as those who were homozygous for the common allele or those who were heterozygous (SULT1A1*1/*2). Among patients who did not receive tamoxifen, there was no association between survival and SULTIAI genotype (HR = 0.7, 95% CI = 0.3 to 1.5). Conclusions: Sulfation of 4-OH TAM provides a previously unanticipated benefit, possibly due to alterations in the bio-availability of the active metabolite or to undefined estrogen receptor-mediated events. These data alternatively suggest that variability in the metabolism of tamoxifen may affect its efficacy.

L131 ANSWER 11 OF 109 SCISEARCH COPYRIGHT 2003 ISI (R)

AB 1. Sulphotransferases are a superfamily of enzymes involved in both detoxification and bioactivation of endogenous and exogenous compounds. The arylsulphotransferase SULT1A1 has been implicated in a decreased activity and thermostability when the wild-type arginine at position 213 of the coding sequence is substituted by a histidine. SULT1A1 is the isoform primarily associated with the conversion of dietary N -OH arylamines to DNA binding adducts and is therefore of interest to determine whether this polymorphism is linked to colorectal **cancer**.

2. Genotyping, using polymerase chain reaction-restriction fragment length polymorphism (PCR-RFLP) analysis, was performed using DNA samples of healthy control subjects (n = 402) and patients with histologically proven colorectal **cancer** (n = 383). Both control and test populations possessed similar frequencies for the mutant allele (32.1 and 31%, respectively; P = 0.935). Results were not altered when age and gender were considered as potential confounders in a logistic regression analysis.

3. Examination of the sulphonating ability of the two allozymes with respect to the substrates p -nitrophenol and paracetamol showed that the affinity and rate of sulphonation was unaffected by substitution of arginine to histidine at position 213 of the amino acid sequence.

4. From this study, we conclude that the SULT1A1 R213H polymorphism is not linked with colorectal **cancer** in this elderly Australian population.

L131 ANSWER 12 OF 109 SCISEARCH COPYRIGHT 2003 ISI (R)DUPLICATE 1

AB The estrogen-signaling pathway plays an important role in the pathophysiology of breast **cancer**, and the sulfoC transferase 1A (SULT1A) family has been found to be both downstream targets of tamoxifen and a **risk** factor of breast **cancer**. We have used PCR-RFLP and direct sequencing methods to determine SULT1A2 polymorph isms in 230 Taiwanese breast **cancer** patients. The results showed that the frequencies of SULT1A2*1 and SULT1A2*2 occurring were 94.8% and 5.2%, respectively. No SULT1A2*3 allele was found in these patients. In comparison with the frequency of healthy controls (96.0% and 4.0% for SULT1A2*1 and SULT1A2*2, respectively), the allelic frequencies of SULT1A2 polymorphisms in these patients were not statistically significant (p = 0.398). However, the SULT1A2*2 allele seems to influence the age of onset among early-onset breast **cancer** patients (p = 0.021, OR = 2.74, 95% CI = 1.13-6.65).

L131 ANSWER 14 OF 109 MEDLINE DUPLICATE 3

AB Carcinogenic aromatic amines such as 4-aminobiphenyl, which is contained in tobacco smoke, are one of the causal factors of urothelial epithelial cancers. 4-Aminobiphenyl has been shown to be bioactivated through N-hydroxylation by hepatic cytochrome (CYP) 1A2 and subsequently through O-sulfation and O-acetylation by **phenol** sulfating **sulfotransferase**, ST1A3 (SULT1A1), and arylamine N-acetyltransferase, NAT2, respectively. In a case-control study for urothelial epithelial cancers, low activity alleles of NAT2 are overall high-**risk** alleles (OR 2.11; 95% CI 1.08-4.26). Wild-type ST1A3*1 ((213)Arg) alleles were slightly overrepresented in nonsmoking urothelial

cancer patients (82.6% vs. 69.7%) and in smoking **cancer** patients (76.7% and 74.3%) compared to a variant ST1A3*2 ((213)His) allele. In combination of ST1A3 and NAT2 genotypes for analyses of urothelial **cancer risk**, the highest OR of 2.45 (95% CI 1.04-5.98) was obtained with ST1A3*1 and NAT2 slow genotype among the 4 combinations. Recombinant ST1A3*1 enzyme showed a tendency of catalyzing higher in vitro 3'-phosphoadenosine 5'-phosphosulfate-dependent DNA adduct formation than ST1A3*2 (2.84 +/- 0.49 and 2.22 +/- 0.11 adducts/10(8) nucleotides). Combined analyses of different alleles of carcinogenic aromatic amine-activating phase II enzymes were applied to urothelial **cancer risk** for the first time and showed the highest **risk** combination of ST1A3 and NAT2 alleles.

L131 ANSWER 19 OF 109 SCISEARCH COPYRIGHT 2003 ISI (R)

AB SULT1A1 enzyme is a member of the sulfotransferase family that alters biological activities of numerous carcinogenic and mutagenic compounds through sulfation. A genetic polymorphism in the coding region of SULT1A1 gene has been associated with modulated enzyme activity. There is a G-->A nucleotide polymorphism in SULT1A1 gene that codes for an Arg-->His substitution, which results in decreased activity and thermal stability of the SULT1A1 enzyme. Utilizing a case-control study design, we hypothesized that the variant allele of the SULT1A1 gene may be associated with lung **cancer risk**. The PCR-RFLP assay was used to successfully genotype the SULT1A1*2 allele (variant A-allele) in 463 Caucasian lung **cancer** cases and 485 frequency matched Caucasian controls. There was an overall significant difference between cases and controls when adjusted by sex and smoking status (adjusted OR = 1.41, 95% CI: 1.04-1.91), The adjusted OR was higher for females (OR = 1.64 95% Cl: 1.06-2.56) than for males (OR = 1.23, 95% CI: 0.80-1.88). Furthermore. the **risk** was significantly higher in current smokers (OR = 1.74, 95% CI: 1.08-2.29) and heavy smokers (OR = 1.45, 95% CI: 1.05-2.00). Our results support the hypothesis that a genetic polymorphism in the SULT1A1 gene may be associated with increased lung **cancer risk**
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L131 ANSWER 24 OF 109 HCAPLUS COPYRIGHT 2003 ACS

AB Sulphation is an important detoxification pathway for numerous xenobiotics; however, it also plays an important role in the metab. and bioactivation of many dietary and environmental mutagens, including heterocyclic amines implicated in the pathogenesis of colorectal and other cancers. A major sulfotransferase (SULT) enzyme in humans, SULT1A1, is polymorphic with the most common variant allele, SULT1A1*2, occurring at a frequency of about 32% in the Caucasian population. This allele codes for an allozyme with low enzyme activity and stability compared to the wild-type (SULT1A1*1) enzyme, and therefore SULT1A1 genotype may influence susceptibility to mutagenicity following exposure to heterocyclic amines and other environmental toxins. Previously, a significant assocn. of SULT1A1*1 genotype with old age was obsd., suggesting a 'chemoprotective' role for the high-activity phenotype. Here the authors have compared the frequencies of the most common SULT1A1 alleles in 226 colorectal **cancer** patients and 293 previously described control patients. We also assessed whether SULT1A1 genotype was related to various clin. parameters in the patient group, including Duke's classification, differentiation, site, nodal involvement and survival. There was no significant difference in allele frequency between the control and **cancer** patient populations, nor was there a significant assocn. with any of the clin. parameters studied. However, when the age-related difference in allele frequency was considered, a significantly reduced **risk** of colorectal **cancer** (odds ratio = 0.47; 95% confidence interval = 0.27-0.83; P=0.009), was assocd. with homozygosity for SULT1A1*1 in subjects under the age of 80 yr. These results suggest that the high activity SULT1A1*1 allozyme protects against dietary and/or environmental chems. involved in the pathogenesis of colorectal

cancer.

L131 ANSWER 26 OF 109 MEDLINE DUPLICATE 8

AB Sulfate conjugation is an important pathway in the metabolism of many drugs, xenobiotic compounds, and hormones. Sulfotransferases (SULTs) catalyze these reactions and have been detected and characterized in various human tissues including the liver and small intestine. Substrates for SULTs that include estrogen and thyroid hormones have well-established roles affecting skeletal integrity and disease processes. We performed the following studies to determine the presence of SULTs in human osteoblast-like cells, and to compare their characteristics to SULTs expressed in other human tissues. Four osteosarcoma cell lines (SaOS-2, U2-OS, PR, and HOS-TE85) were screened for the presence of four different SULT activities. Predominant activities were found for SULT1A1 in SaOS-2 cells, and SULT-1A3 in HOS-TE85 cells. Several biochemical properties of each enzyme that included apparent K(m) values, thermal stabilities, and responses to the inhibitors 2,6-dichloro-4-nitrophenol and NaCl were used to further characterize the SULT activities. High-performance liquid chromatography (HPLC) of the reaction products confirmed the known products of SULT1A1 and SULT1A3. When the mature human osteoblast HOB-03-CE6 cell line was tested for activity alone, the predominant activity was SULT1A3, with minimal SULT1A1. The results indicate that SULT1A1 and SULT1A3 are present in human osteosarcoma and mature osteoblast cell lines, and that the characteristics of the osteosarcoma cell SULTs are similar to those expressed in other human tissues. SULTs may have regulatory roles in the deactivation of thyroid hormones or estrogenic compounds in bone, and thus may affect hormone action and bone responses in the human skeleton.

L131 ANSWER 33 OF 109 MEDLINE DUPLICATE 10

AB In recent years, significant effort has been made to identify genes that influence breast **cancer risk**. Because the high-penetrance breast **cancer** susceptibility genes BRCA1 and 2 play a role only in a small fraction of breast **cancer** cases, understanding the genetic **risk** of the majority of breast cancers will require the identification and analysis of several lower penetrance genes. The estrogen-signaling pathway plays a crucial role in the pathophysiology of breast **cancer**; therefore, polymorphism in genes involved in this pathway is likely to influence breast **cancer risk**. Our detailed analysis of gene expression profiles of estrogen- and 4-OH-tamoxifen-treated ZR75-1 breast **cancer** cells identified members of the **sulfotransferase** 1A (SULT1A) **phenol sulfotransferase** family as downstream targets of tamoxifen. On the basis of the induction of SULT1A by 4-OH-tamoxifen and the known inherited variability in SULT1A enzymatic activity, we hypothesized that polymorphism in sulfotransferase genes might influence the **risk** of breast **cancer**. Using an RFLP that distinguishes an arginine to histidine change in exon 7 of the SULT1A1 gene, we characterized SULT1A1 genotypes in relation to breast **cancer risk**. An analysis of 444 breast **cancer** patients and 227 controls revealed no effect of SULT1A1 genotype on the **risk** of breast **cancer** (P = 0.69); however, it did appear to influence the age of onset among early-onset affected patients (P = 0.04). Moreover, individuals with the higher activity SULT1A1*1 allele were more likely to have other **tumors** in addition to breast **cancer** (P = 0.004; odds ratio, 3.02; 95% confidence interval, 1.32, 8.09). The large number of environmental mutagens and carcinogens activated by sulfotransferases and the high frequency of the SULT1A1*1 allele in human populations warrants additional studies to address the role of SULT genes in human **cancer**.

L131 ANSWER 37 OF 109 BIOTECHDS COPYRIGHT 2003 THOMSON DERWENT AND ISI

AB The relationship of aryl-sulfotransferase (EC-2.8.2.1) activity (SULT1A1) genotype to sulfotransferase phenotype in platelet cytosol was evaluated.

DNA was obtained from blood samples taken from participants which include those diagnosed with incident, primary and histologically confirmed **cancer** of the colon/rectum or mamma, as well as community control subjects. The region of the SULT1A1-1 gene flanking the polymorphic bp (G to A transition) was amplified in a polymerase chain reaction (PCR) using forward and reverse DNA primers. Comparison of the DNA primers, using the NCBI BLAT database, showed no significant homology with either SULT1A2 or SULT1A3. The resulting PCR product of 281 bp were resolved on a 3% agarose gel and visualized by ethidium bromide staining and UV transillumination. Confirmation of the genotypes were performed by direct sequencing of representative samples using a 33P radiolabel sequencing kit. A simple colorimetric phenotyping assay, in conjunction with genotyping, was used to show that there was a significant correlation SULT1A1 genotype and platelet sulfotransferase activity towards 2-napthol, a marker substrate. (61 ref)

L131 ANSWER 45 OF 109 LIFESCI COPYRIGHT 2003 CSA DUPLICATE 15

AB Preliminary evidence suggests that genetic polymorphisms in certain enzymes involved in xenobiotic metabolism and chemical defense could modify a susceptibility to prostate **cancer**. In the present study, two recently described phenol sulphotransferaseSULT1A1 alleles (SULT1A1*1,SULT1A1*2) were investigated using a polymerase chain reaction-restriction fragment length polymorphism (PCR-RFLP) approach. Genotyping was performed on DNA isolated from white blood cells from 134 patients with prostate **cancer** and 184 healthy control subjects. Both the prostate **cancer** patients and the controls demonstrated similar frequencies of the variant alleleSULT1A1*2 (35.1% vs 39.1%). Homozygosity for the variant allele was slightly less frequent in **cancer** patients than controls (12.7% vs 17.4%). Our study does not support the hypothesis that the phenol sulphotransferase variant alleleSULT1A1*2 with a G/A transition at nucleotide 638 is a **risk** modifier for prostate **cancer** in the Caucasian population.

L131 ANSWER 48 OF 109 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.

L131 ANSWER 65 OF 109 MEDLINE DUPLICATE 21

AB It has become clear that several polymorphisms of human drug-metabolizing enzymes influence an individual's susceptibility for chemical carcinogenesis. This review gives an overview on relevant polymorphisms of four families of drug-metabolizing enzymes. Rapid acetylators (with respect to N-acetyltransferase NAT2) were shown to have an increased **risk** of colon **cancer**, but a decreased **risk** of bladder **cancer**. In addition an association between a NAT1 variant allele (NAT*10, due to mutations in the polyadenylation site causing approximately two fold higher activity) and colorectal **cancer** among NAT2 rapid acetylators was observed, suggesting a possible interaction between NAT1 and NAT2. Glutathione S-transferases M1 and T1 (GSTM1 and GSTT1) are polymorphic due to large deletions in the structural gene. Meta-analysis of 12 case-control studies demonstrated a significant association between the homozygous deletion of GSTM1 (GSTM1-0) and lung **cancer** (odds ratio: 1.41; 95% CI: 1.23-1.61). Combination of GSTM1-0 with two allelic variants of cytochrome P4501A1 (CYP1A1), CYP1A1 m2/m2 and CYP1A1 Val/Val further increases the **risk** for lung **cancer**. Indirect mechanisms by which deletion of GSTM1 increases **risk** for lung **cancer** may include GSTM1-0 associated decreased expression of GST M3 and increased activity of CYP1A1 and 1A2. Combination of GST M1-0 and NAT2 slow acetylation was associated with markedly increased **risk** for lung **cancer** (odds ratio: 7.8; 95% CI: 1.4-78.7). In addition GSTM1-0 is clearly associated with bladder **cancer** and possibly also with colorectal, hepatocellular, gastric, esophageal (interaction with CYP1A1), head and neck as well as cutaneous **cancer**. In individuals with the GSTT1-0 genotype more chromosomal aberrations and sister chromatid exchanges (SCEs) were observed after exposure to 1,3-butadiene or various

haloalkanes or haloalkenes. Evidence for an association between GSTT1-0 and myelodysplastic syndrome and acute lymphoblastic leukemia has been presented. A polymorphic site of GSTP1 (valine to isoleucine at codon 104) decreases activity to several carcinogenic diol epoxides and was associated with testicular, bladder and lung **cancer**. Microsomal expoxide hydrolase (mEH) is polymorphic due to amino acid variation at residues 113 and 139. Polymorphic variants of mEH were associated with hepatocellular **cancer** (His-113 allele), ovarian **cancer** (Tyr-113 allele) and chronic obstructive pulmonary disease (His-113 allele). Three human **sulfotransferases** (STs) are regulated by genetic polymorphisms (hDHEAST, hM-**PST**, TS **PST**). Since a large number of environmental mutagens are activated by STs an association with human **cancer risk** might be expected.

L131 ANSWER 66 OF 109 CANCERLIT

AB Metabolic pathways leading to carcinogen activation and inactivation appear to contribute to human **cancer** susceptibility. Phenolsulfotransferases represent an important family of enzymes for further study, since they are thought to mediate both bioactivation and detoxification reactions in experimental animals. The thermostable form of phenolsulfotransferase (TS-**PST**; ST1A3), which is polymorphic in humans, catalyzes the sulfation of important environmental carcinogens (e.g., N-hydroxy arylamines and N-hydroxy heterocyclic amines). However, its role in the development of specific endogenous human cancers is unknown. We have developed a simple and reproducible microtiter-plate colorimetric method for measurement of TS-**PST** activity, and have validated its use in a large epidemiological case-control study of colon **cancer**. Human platelet cytosols were used since platelet TS-**PST** activity was previously shown to correlate with human liver and colon TS-**PST** activity. Here we report that **sulfotransferase** (ST1A3, as measured by 2-naphthol activity) appears as a potential protective factor for colon **cancer**. The population (n=221) segregated into two distinct phenotypes based on TS-**PST** activity: 'fast' and 'slow'. However, the slow phenotype was more frequently associated with colon **cancer** than with controls (56% vs 40%, p=0.03), suggesting that TS-**PST** should be included in future models of **cancer risk**.

L131 ANSWER 70 OF 109 SCISEARCH COPYRIGHT 2003 ISI (R)DUPLICATE 24

AB Gene-environment interaction is an important aspect of human **cancer risk**. Genetic polymorphisms in acetylation and N-oxidation have previously been described regarding their impact on the heterocyclic amine-induced **risk** for colon **cancer**. Here, we report that another enzyme involved in the metabolism of food-borne carcinogens, **sulfotransferase** (ST1A3 measured by 2-naphthol activity), may function as a potential protective factor for colon **cancer** in humans. Initially characterized in human liver and colon (Chou et al., 1995), TS-**PST** activity can also be measured in platelets. A simple microtiter-based colorimetric technique was developed for use in this case-control study. African-Americans had a higher mean ST activity than Caucasians (2.32 +/- 0.24 versus 1.77 +/- 0.09 nmols/min per mg cytosolic protein, P = 0.036). Furthermore, the slow ST phenotype (ST less than or equal to 1.53) was more frequently associated with colon **cancer** than controls (57 versus 40%, P = 0.026). These data suggest that the ST1A3 isoform may play a role in the differential **risk** for colorectal **cancer**. (C) 1997 Elsevier Science B.V.

L131 ANSWER 79 OF 109 CANCERLIT

AB Sulfate conjugation is an important pathway in the biotransformation of drugs, xenobiotic compounds, hormones and neurotransmitters. Sulfation usually results in termination of the biological activity of a compound, since it increases water solubility and, therefore, renal excretion. In some cases, however, sulfate conjugation can result in the bioactivation

of drugs or procarcinogens. For that reason, and also because sulfation is an important pathway in the metabolism of steroid hormones, differences among individuals in the ability to catalyze this reaction could result in individual variation in susceptibility to chemically induced neoplasia or in the pathophysiology of steroid hormone-responsive **tumors**.
Before discussing variation among individuals in sulfation, it will be necessary to review briefly our current understanding of the biochemistry and molecular biology of enzymes that catalyze sulfate conjugation in humans. The **sulfotransferase** (ST) enzymes which catalyze the sulfate conjugation of drugs, xenobiotic compounds, hormones and neurotransmitters in mammals are cytosolic homodimers with monomer mol wt values that vary from 30 to 35 kD. The sulfate donor for the reactions catalyzed by these enzymes is 3'-phosphoadenosine-5'-phosphosulfate (PAPS). cDNA sequences for 19 cytosolic ST enzymes have been reported to this time. Four of those cDNAs encode enzymes that are expressed in human tissue. The dendrogram shown in a figure depicts relationships among the amino acid sequences encoded by those 19 cDNAs. It shows that, on the basis of primary amino acid sequence, cytosolic ST enzymes can be classified as members of three 'families,' one of which includes two 'subfamilies.' Families include proteins with 45% or greater sequence identity and consist of phenol STs (PSTs), hydroxysteroid STs (HSSTs), and the flavonol STs (FSTs) that are expressed in plants. The **PST** family includes two subfamilies with 60% or greater amino acid sequence identity, the estrogen STs (ESTs) and PSTs. Two forms of **PST** are present in human tissues, a 'thermolabile' (TL) or 'monoamine' (M) form that preferentially catalyzes the sulfate conjugation of monoamine neurotransmitters such as dopamine, and a 'thermostable' (TS) or 'phenol' (P) form that preferentially catalyzes the sulfation of simple planar phenols such as 4-nitrophenol. An EST cDNA with a very low Km value for estrone has also been cloned from human liver. The only member of the HSST family characterized in human tissue to this time is referred to most often as dehydroepiandrosterone (DHEA) ST. All of these human enzymes have been expressed in either bacterial or mammalian systems, and the biochemical characteristics of the expressed enzymes have been studied. The gene for DHEA ST in humans is located on chromosome 19q13.3, while the genes for both forms of **PST** are localized to chromosome 16p11-12. The human EST gene is on chromosome 4. Even though they are located on three different chromosomes, the gene structures of human EST, DHEA ST, and TL **PST** show significant structural homology.
Measurements of enzymatic activity have demonstrated large individual variations in levels of TS **PST**, TL **PST** and DHEA ST activities in human tissues. Furthermore, variations in TS **PST** and TL **PST** activities in an easily accessible human tissue, the platelet, are controlled by separate genetic polymorphisms. The possibility of genetic regulation of DHEA ST activity has not been explored with family studies or segregation analysis, but the level of this enzyme activity in human liver shows a bimodal frequency distribution, with approximately 20-25% of tissue samples included in a high activity subgroup. These observations raise the possibility that this ST activity, like those of TS and TL **PST** in human tissue, might also be regulated by a genetic polymorphism. Increasing understanding of the biochemistry, molecular biology and 'pharmacogenetics' of ST enzymes will now make it possible to study the role of differences in these enzyme activities among individuals and ethnic group(ABSTRACT TRUNCATED)

L131 ANSWER 97 OF 109 CANCERLIT

AB Xenobiotics continue to be found that are metabolized in mammals to electrophilic reactants that form covalently bound adducts in cellular DNA leading to **tumor** formation. This discussion will be limited to (A). Sulfonation (sulfation) and its inhibition in chemical carcinogenesis, and (B). Etheno-base adducts in presumed untreated rats and mice. (A) Water-stable sulfates have long been known as cell components and as detoxification products of many xenobiotics. These sulfates arise from the activities of sulfotransferases with the cofactor

3'-phosphoadenosine-5'-phosphosulfate (PAPS). Recently, this sulfonation system has been found to be a major route of activation of several classes of carcinogens by the formation of toxic and water-labile (short half-life) sulfuric acid esters. Heterolytic cleavage of these esters generates sulfate ions and highly reactive mutagenic and carcinogenic electrophiles that adduct DNA. These sulfonations can be inhibited by pentachlorophenol (PCP; for **phenol sulfotransferases**) or by dehydroepiandrosterone (DHEA; for hydroxysteroid sulfotransferases). Brachymorphic mice are useful test animals; these mice have genetic impairments of the synthesis of PAPS. Activation by sulfonation was first noted in the complex metabolism of 2-acetylaminofluorene. Recently, sulfonation of the metabolite N-hydroxy-2-aminofluorene was noted to form 2-aminofluorene-N-sulfate, a very reactive electrophile that forms only one DNA adduct, N-(deoxyguanosin-8-yl)-2-aminofluorene in mouse liver. Pretreatment with PCP greatly inhibits DNA adduct and hepatoma formation. The natural allylic benzenes safrole and estragole are oxidized by Cyt P-450 to proximate carcinogenic 1'-hydroxy metabolites. Sulfonation of these compounds generates sulfooxy ultimate carcinogens that form multiple adducts in the liver DNA of mice. PCP inhibits the formation of these adducts and liver **tumors**. The synthetic acetylenic 1'-hydroxy-2',3'-dehydro analogs of the 1'-hydroxy allylic metabolites are much more carcinogenic and its O-sulfate forms only deoxyguanosin-N'-yl adducts in the liver DNA. Hydroxymethyl polycyclic hydrocarbons are sulfonated to form electrophilic sulfooxy metabolites that react with each of the base amino groups in DNA to form benzylic adducts. This sulfonation is inhibited by DHEA but not by PCP. 6-Sulfooxymethyl-benzo(a)pyrene is very hepatocarcinogenic in mice. The carcinogens noted above that form only one DNA adduct may be useful in studies on the mutagenic activation of proto-oncogenes and suppressor genes. (B) Some carcinogens may be derived endogenously and form DNA adducts in vivo. N2,3-ethenoguanine, found after depurination of the liver DNA from presumed untreated rats (Fedtke et al, 1990), may be an example. Similarly, we have noted in unpublished studies the formation of low levels of 1,N6-etheno-adenosine by liver microsomes of presumed untreated mice after incubation with NADPH, oxygen, and adenosine. The precursors of these etheno bases are not known at present.

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COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	304.64	304.85

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE ENTRY	TOTAL SESSION
CA SUBSCRIBER PRICE	-0.65	-0.65

STN INTERNATIONAL LOGOFF AT 10:32:55 ON 10 APR 2003

	L #	Hits	Search Text	DBs	Time Stamp
1	L1	36	sulfotransferase$1 NEAR5 PHENOL$8	USPAT; US-PGPUB	2003/04/10 09:40
2	L2	20	sulfotransferase$1 AND PST	USPAT; US-PGPUB	2003/04/10 09:40
3	L3	46	1 or 2	USPAT; US-PGPUB	2003/04/10 09:40

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TITLE: Individualization of therapy with immunosuppressants

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INVENTOR-INFORMATION:

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APPL-NO: 10/ 100556

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RELATED-US-APPL-DATA:

non-provisional-of-provisional 60275489 20010314 US

US-CL-CURRENT: 424/9.2

ABSTRACT:

The invention relates to the individualization of therapy on the basis of a phenotypic profile of an individual. More specifically, the present invention relates to the use of metabolic phenotyping for the individualization of treatment with immunosuppressants.

RELATED APPLICATION

[0001] This application is a new application which claims the benefit of U.S. Provisional Application No. 60/275,489, filed on Mar. 14, 2001. The entire teachings of the above application is incorporated herein by reference.

---------- KWIC ---------

Summary of Invention Paragraph - BSTX (5):

[0005] For the majority of drugs (or xenobiotics) administered to humans, their fate is to be metabolized in the liver, into a form less toxic and lipophilic with their subsequent excretion in the urine. Their metabolism involves two systems (Phase I and Phase II) which act consecutively: Phase I enzymes include the cytochrome P450 system which includes at least 20 enzymes catalyzing oxidation reactions as well as carboxylesterase, amindases, epoxide hydrolase, quinine reductase, alcohol and aldehyde dehydrogenase, xanthine oxidase and flavin-containing monooxygenase. These enzymes are localized in

the microsomal fraction. Phase II enzymes include the conjugation system which involves at least 5 enzymes including, N-acetyltransferases (NAT), UDP-glucoronyltransferases (UGT), **sulfotransferases** (SUT), and glutathione-S-transferases (GST). A detailed description of the complex human drug metabolizing systems is provided in Kumar and Surapaneni (Medicinal Res. Rev. (2001) 21(5):397-411) and patent application WO 01/59127 A2.

Summary of Invention Paragraph - BSTX (10):

[0010] DMEs have been classified according to the type of reaction they catalyze and the cofactors involved. The major classes of Phase I enzymes include, but are not limited to, cytochrome P450 and flavin-containing monooxygenase. Other enzyme classes involved in Phase 1-type catalytic cycles and reactions include, but are not limited to, NADPH cytochrome P450 reductase (CPR), the microsomal cytochrome b5/NADH cytochrome b5 reductase system, the ferredoxin/ferredoxin reductase redox pair, aldo/keto reductases, and alcohol dehydrogenases. The major classes of Phase II enzymes include, but are not limited to, UDP glucuronyltransferase, **sulfotransferase,** glutathione S-transferase, N-acyltransferase, and N-acetyl transferase.

Summary of Invention Paragraph - BSTX (48):

[0048] **Sulfotransferase**

Summary of Invention Paragraph - BSTX (49):

[0049] Sulfate conjugation occurs on many of the same substrates which undergo O-glucuronidation to produce a highly water-soluble sulfuric acid ester. **Sulfotransferases** (ST) catalyze this reaction by transferring SO.sub.3-from the cofactor 3'-phosphoadenosine-5'-phosphosulfate (PAPS) to the substrate. ST substrates are predominantly phenols and aliphatic alcohols, but also include aromatic amines and aliphatic amines, which are conjugated to produce the corresponding sulfamates. The products of these reactions are excreted mainly in urine.

Summary of Invention Paragraph - BSTX (50):

[0050] STs are found in a wide range of tissues, including liver, kidney, intestinal tract, lung, platelets, and brain. The enzymes are generally cytosolic, and multiple forms are often co-expressed. For example, there are more than a dozen forms of ST in rat liver cytosol. These biochemically characterized STs fall into five classes based on their substrate preference: arylsulfotransferase, alcohol **sulfotransferase,** estrogen **sulfotransferase,** tyrosine ester **sulfotransferase,** and bile salt **sulfotransferase**.

Summary of Invention Paragraph - BSTX (52):

[0052] Several forms of ST have been purified from human liver cytosol and

cloned. There are two **phenol sulfotransferases** with different thermal stabilities and substrate preferences. The thermostable enzyme catalyzes the sulfation of phenols such as para-nitrophenol, minoxidil, and acetaminophen; the thermolabile enzyme prefers monoamine substrates such as dopamine, epinephrine, and levadopa. Other cloned STs include an estrogen **sulfotransferase** and an N-acetylglucosamine-6-O-**sulfotransferase**-. This last enzyme is illustrative of the other major role of STs in cellular biochemistry, the modification of carbohydrate structures that may be important in cellular differentiation and maturation of proteoglycans. Indeed, an inherited defect in a **sulfotransferase** has been implicated in macular corneal dystrophy, a disorder characterized by a failure to synthesize mature keratan sulfate proteoglycans (Nakazawa, K. et al. (1984) J. Biol. Chem. 259: 13751-7).

Detail Description Paragraph - DETX (218):

[0391] As mentioned previously the CYP2E1 gene has multiple polymorphisms. An example of a procedure for genotyping CYP2E1 for the most common mutations, those termed the **Pst**/Rsal and Dral mutations (allows genotyping of CYP2E1*5A, CYP2E1*5B and CYP2E1*6), involves the amplification of a fragment containing either the Pstl and Rsal restriction sites or the Dral restriction site using specific primers (Nedelcheva et al. (1996) Methods in Enzymology 272:218-225). The amplified product is then incubated with the appropriate restriction enzyme (Pstl or Rsal/Dral) and the digestion products separated electrophoretically. From an allele with wt sequence at the Pstl or Rsal site, the 510 bp fragment produced by PCR is cleaved to a 360 bp and a 150 bp fragment. From the mutant allele the 510 bp fragment remains uncleaved. From an allele with the wt sequence at the Dral mutation site, the 370 bp PCR amplified fragment is cleaved to a 240 bp and 130 bp pair of fragments, while the mutant allele is uncleaved.

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INVENTOR-INFORMATION:

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ABSTRACT:

The invention relates to the individualization of therapy on the basis of a phenotypic profile of an individual. More specifically, the present invention relates to the use of metabolic phenotyping for the individualization of treatment with hyperlipidemia agents.

RELATED APPLICATION

[0001] This application is a new application which claims the benefit of U.S. Provisional Application No. 60/284,210, filed on Apr. 18, 2001. The entire teachings of the above application is incorporated herein by reference.

---------- KWIC ---------

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Summary of Invention Paragraph - BSTX (52):

[0052] Several forms of ST have been purified from human liver cytosol and

cloned. There are two **phenol sulfotransferases** with different thermal stabilities and substrate preferences. The thermostable enzyme catalyzes the sulfation of phenols such as para-nitrophenol, minoxidil, and acetaminophen; the thermolabile enzyme prefers monoamine substrates such as dopamine, epinephrine, and levadopa. Other cloned STs include an estrogen **sulfotransferase** and an N-acetylglucosamine-6-O-**sulfotransferase**- . This last enzyme is illustrative of the other major role of STs in cellular biochemistry, the modification of carbohydrate structures that may be important in cellular differentiation and maturation of proteoglycans. Indeed, an inherited defect in a **sulfotransferase** has been implicated in macular corneal dystrophy, a disorder characterized by a failure to synthesize mature keratan sulfate proteoglycans (Nakazawa, K. et al. (1984) J. Biol. Chem. 259: 13751-7).

Detail Description Paragraph - DETX (245):

[0368] As mentioned previously the CYP2E1 gene has multiple polymorphisms. An example of a procedure for genotyping CYP2E1 for the most common mutations, those termed the **Pst**/Rsal and Dral mutations (allows genotyping of CYP2E1*5A, CYP2E1*5B and CYP2E1*6), involves the amplification of a fragment containing either the Pstl and Rsal restriction sites or the Dral restriction site using specific primers (Nedelcheva et al. (1996) Methods in Enzymology 272:218-225). The amplified product is then incubated with the appropriate restriction enzyme (Pstl or Rsal/Dral) and the digestion products separated electrophoretically. From an allele with wt sequence at the Pstl or Rsal site, the 510 bp fragment produced by PCR is cleaved to a 360 bp and a 150 bp fragment. From the mutant allele the 510 bp fragment remains uncleaved. From an allele with the wt sequence at the Dral mutation site, the 370 bp PCR amplified fragment is cleaved to a 240 bp and 130 bp pair of fragments, while the mutant allele is uncleaved.

PGPUB-DOCUMENT-NUMBER: 20030050232

PGPUB-FILING-TYPE: new

DOCUMENT-IDENTIFIER: US 20030050232 A1

TITLE: Novel human proteins, polynucleotides encoding them and methods of using the same

PUBLICATION-DATE: March 13, 2003

INVENTOR-INFORMATION:

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APPL-NO: 09/ 839446

DATE FILED: April 19, 2001

RELATED-US-APPL-DATA:

non-provisional-of-provisional 60198293 20000419 US

non-provisional-of-provisional 60198645 20000420 US

non-provisional-of-provisional 60210809 20000609 US

non-provisional-of-provisional 60199476 20000425 US

non-provisional-of-provisional 60200025 20000426 US

non-provisional-of-provisional 60224610 20000811 US

non-provisional-of-provisional 60200024 20000426 US

non-provisional-of-provisional 60199880 20000426 US

non-provisional-of-provisional 60218591 20000717 US

non-provisional-of-provisional 60271814 20010227 US

US-CL-CURRENT: 514/12, 435/189 , 435/320.1 , 435/325 , 435/69.1 , 536/23.2

ABSTRACT:

The invention provides polypeptides, designated herein as POLYX polypeptides, as well as polynucleotides encoding POLYX polypeptides, and antibodies that immunospecifically-bind to POLYX polypeptide or polynucleotide, or derivatives, variants, mutants, or fragments thereof. The invention additionally provides methods in which the POLYX polypeptide, polynucleotide, and antibody are used in the detection, prevention, and treatment of a broad range of pathological states.

RELATED APPLICATIONS

[0001] This application claims priority to USS No. 60/198,293(15966-776), filed Apr. 19, 2000; No. 60/198,645 (15966-777), filed Apr. 20, 2000; No. 60/210,809 (15966-778A), filed Jun. 9, 2000; No. 60/199,476 (15966-778), filed Apr. 26, 2000; No. 60/200,025 (15966-779), filed Apr. 26, 2000; No. 60/224,610 (15966-780A), filed Aug. 11, 2000; No. 60/200,024 (15966-780), filed Apr. 26, 2000; No. 60/199,880 (15966-781) filed Apr. 26, 2000; No. 60/218,591(21402-059), filed Jul. 17, 2000; and No. 60/271,814 (21402-059A), filed Feb. 27, 2001. The contents of this application are incorporated by reference in its entirety.

---------- KWIC ---------

Summary of Invention Paragraph - BSTX (148):

[0145] Sulfation is an important pathway in the biotransformation of steroid hormones such as estrogens. Human liver contains two different types of **sulfotransferases, dehydroepiandrosterone (DHEA) sulfotransferase and phenol sulfotransferase**. Estrogen preferring sulfotransferases are cytosolic proteins present in liver, intestine, and in kidney (at lower concentrations). Functionally, the enzyme is believed to control the level of the estrogen receptor by sulfurylating free estradiol. It maximally sulfates beta-estradiol and estrone at concentrations of 20 nm, and dehydroepiandrosterone, pregnenolone, ethinylestradiol, equalenin, diethylstilbesterol, and 1-naphthol at significantly higher concentrations. However, cortisol, testosterone, and dopamine are not sulfated by the estrogen preferring sulfotransferases.

PGPUB-DOCUMENT-NUMBER: 20030049204

PGPUB-FILING-TYPE: new

DOCUMENT-IDENTIFIER: US 20030049204 A1

TITLE: Individualization of therapy with gastroesophageal reflux disease agents

PUBLICATION-DATE: March 13, 2003

INVENTOR-INFORMATION:

NAME	CITY	STATE	COUNTRY	RULE-47
Leyland-Jones, Brian	Miami	FL	US	

APPL-NO: 10/ 132080

DATE FILED: April 24, 2002

RELATED-US-APPL-DATA:

non-provisional-of-provisional 60285687 20010424 US

US-CL-CURRENT: 424/9.1, 435/7.92

ABSTRACT:

The invention relates to the individualization of therapy on the basis of a phenotypic profile of an individual. More specifically, the present invention relates to the use of metabolic phenotyping for the individualization of treatment with GERD agents.

RELATED APPLICATION

[0001] This application is a new application which claims the benefit of U.S. Provisional Application No. 60/285,687, filed on Apr. 24, 2001. The entire teachings of the above application is incorporated herein by reference.

---------- KWIC ---------

Summary of Invention Paragraph - BSTX (3):

[0003] For the majority of drugs (or xenobiotics) administered to humans, their fate is to be metabolized in the liver, into a form less toxic and lipophilic with their subsequent excretion in the urine. Their metabolism involves two systems (Phase I and Phase II) which act consecutively: Phase I enzymes include the cytochrome P450 system which includes at least 20 enzymes catalyzing oxidation reactions as well as carboxylesterase, amindases, epoxide hydrolase, quinine reductase, alcohol and aldehyde dehydrogenase, xanthine

oxidase and flavin-containing monooxygenase. These enzymes are localized in the microsomal fraction. Phase II enzymes include the conjugation system which involves at least 5 enzymes including, N-acetyltransferases (NAT), UDP-glucoronyltransferases (UGT), **sulfotransferases** (SUT), and glutathione-S-transferases (GST). A detailed description of the complex human drug metabolizing systems is provided in Kumar and Surapaneni (Medicinal Res. Rev. (2001) 21(5):397-411) and patent application WO 01/59127 A2.

Summary of Invention Paragraph - BSTX (8):

[0008] DMEs have been classified according to the type of reaction they catalyze and the cofactors involved. The major classes of Phase I enzymes include, but are not limited to, cytochrome P450 and flavin-containing monooxygenase. Other enzyme classes involved in Phase 1-type catalytic cycles and reactions include, but are not limited to, NADPH cytochrome P450 reductase (CPR), the microsomal cytochrome b5/NADH cytochrome b5 reductase system, the ferredoxin/ferredoxin reductase redox pair, aldo/keto reductases, and alcohol dehydrogenases. The major classes of Phase II enzymes include, but are not limited to, UDP glucuronyltransferase, **sulfotransferase,** glutathione S-transferase, N-acyltransferase, and N-acetyl transferase.

Summary of Invention Paragraph - BSTX (46):

[0046] **Sulfotransferase**

Summary of Invention Paragraph - BSTX (47):

[0047] Sulfate conjugation occurs on many of the same substrates which undergo O-glucuronidation to produce a highly water-soluble sulfuric acid ester. **Sulfotransferases** (ST) catalyze this reaction by transferring SO.sub.3-- from the cofactor 3'-phosphoadenosine-5'-phosphosulfate (PAPS) to the substrate. ST substrates are predominantly phenols and aliphatic alcohols, but also include aromatic amines and aliphatic amines, which are conjugated to produce the corresponding sulfamates. The products of these reactions are excreted mainly in urine.

Summary of Invention Paragraph - BSTX (48):

[0048] STs are found in a wide range of tissues, including liver, kidney, intestinal tract, lung, platelets, and brain. The enzymes are generally cytosolic, and multiple forms are often co-expressed. For example, there are more than a dozen forms of ST in rat liver cytosol. These biochemically characterized STs fall into five classes based on their substrate preference: arylsulfotransferase, alcohol **sulfotransferase,** estrogen **sulfotransferase,** tyrosine ester **sulfotransferase,** and bile salt **sulfotransferase**.

Summary of Invention Paragraph - BSTX (50):

[0050] Several forms of ST have been purified from human liver cytosol and cloned. There are two **phenol sulfotransferases** with different thermal stabilities and substrate preferences. The thermostable enzyme catalyzes the sulfation of phenols such as para-nitrophenol, minoxidil, and acetaminophen; the thermolabile enzyme prefers monoamine substrates such as dopamine, epinephrine, and levadopa. Other cloned STs include an estrogen **sulfotransferase** and an N-acetylglucosamine-6-O**-sulfotransferase**- . This last enzyme is illustrative of the other major role of STs in cellular biochemistry, the modification of carbohydrate structures that may be important in cellular differentiation and maturation of proteoglycans. Indeed, an inherited defect in a **sulfotransferase** has been implicated in macular corneal dystrophy, a disorder characterized by a failure to synthesize mature keratan sulfate proteoglycans (Nakazawa, K. et al. (1984) J. Biol. Chem. 259: 13751-7).

Detail Description Paragraph - DETX (219):

[0379] As mentioned previously the CYP2E1 gene has multiple polymorphisms. An example of a procedure for genotyping CYP2E1 for the most common mutations, those termed the **Pst**/RsaI and DraI mutations (allows genotyping of CYP2E1*5A, CYP2E1*5B and CYP2E1*6), involves the amplification of a fragment containing either the PstI and RsaI restriction sites or the DraI restriction site using specific primers (Nedelcheva et al. (1996) Methods in Enzymology 272:218-225). The amplified product is then incubated with the appropriate restriction enzyme (PstI or RsaI/DraI) and the digestion products separated electrophoretically. From an allele with wt sequence at the PstI or RsaI site, the 510 bp fragment produced by PCR is cleaved to a 360 bp and a 150 bp fragment. From the mutant allele the 510 bp fragment remains uncleaved. From an allele with the wt sequence at the DraI mutation site, the 370 bp PCR amplified fragment is cleaved to a 240 bp and 130 bp pair of fragments, while the mutant allele is uncleaved.

PGPUB-DOCUMENT-NUMBER: 20030008366

PGPUB-FILING-TYPE: new

DOCUMENT-IDENTIFIER: US 20030008366 A1

TITLE: Polypeptide of N-acetylglucosamine-6-O-**sulfotransferase** and DNA encoding the same

PUBLICATION-DATE: January 9, 2003

INVENTOR-INFORMATION:

NAME	CITY	STATE	COUNTRY	RULE-47
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Muramatsu, Takashi	Nagoya-shi		JP	

APPL-NO: 10/ 212933

DATE FILED: August 5, 2002

RELATED-US-APPL-DATA:

child 10212933 A1 20020805

parent division-of 09471867 19991223 US GRANTED

parent-patent 6455289 US

child 09471867 19991223 US

parent division-of 09263023 19990305 US GRANTED

parent-patent 6037159 US

FOREIGN-APPL-PRIORITY-DATA:

COUNTRY	APPL-NO	DOC-ID	APPL-DATE
JP	10-54007	1998JP-10-54007	March 5, 1998
JP	10-177844	1998JP-10-177844	June 24, 1998

US-CL-CURRENT: 435/183, 514/12

ABSTRACT:

Apolypeptide of N-acetylglucosamine-6-O-**sulfotransferase** and a DNA encoding the peptide are provided. The polypeptide is (a) or (b) below:
(a) a polypeptide consisting of an amino acid sequence represented by SEQ ID NO: 2; or

(b) a polypeptide which comprises an amino acid sequence including substitution, deletion, insertion or transposition of one or few amino acids in the amino acid sequence of (a) and which has an enzymatic activity to transfer a sulfate group from a sulfate group donor to a hydroxyl group at 6 position of an N-acetylglucosamine residue located at a non-reducing end of an oligosaccharide represented by the formula I:
GlcNAc.beta.1-3Gal.beta.1-4GlcNAc (I)
wherein GlcNAc represents an N-acetylglucosamine residue, Gal represents a galactose residue, .beta.1-3 represents a .beta.1-3 glycosidic linkage, and .beta.1-4 represents a .beta.1-4 glycosidic linkage.

---------- KWIC ---------

Abstract Paragraph - ABTX (1):

Apolypeptide of N-acetylglucosamine-6-O<u>**-sulfotransferase**</u> and a DNA encoding the peptide are provided. The polypeptide is (a) or (b) below:

Title - TTL (1):

Polypeptide of N-acetylglucosamine-6-O<u>**-sulfotransferase**</u> and DNA encoding the same

Summary of Invention Paragraph - BSTX (2):

[0001] The present invention relates to a polypeptide of N-acetylglucosamine-6-O<u>**-sulfotransferase**</u> and a DNA having a nucleotide sequence encoding the polypeptide.

Summary of Invention Paragraph - BSTX (5):

[0003] It has been described in Biochem. J., 319, 209-216 (1996) and J. Biol. Chem. 272, 29493-29501 (1997) that rat and human microsome fractions had an N-acetylglucosamine-6-O<u>**-sulfotransferase**</u> activity. However, there has been so far no report about isolation and identification of a polypeptide of N-acetylglucosamine-6-O<u>**-sulfotransfera- se**</u>. A DNA encoding this polypeptide has not also been known.

Summary of Invention Paragraph - BSTX (6):

[0004] Once a polypeptide of N-acetylglucosamine-6-O<u>**-sulfotransferase**</u> is obtained, it can be used for synthesis of sugar chains such as GlyCAM-1 that is a ligand of L-selectin (which is involved in homing of lymphocytes and rolling of leukocytes occurring at the early stage of inflammation). A DNA encoding this polypeptide would be expected to be used for large scale production of the polypeptide, or artificial synthesis of GlyCAM-1 (having a structure of NeuAc.alpha.2-3Gal.beta.1-4(- Fuc.alpha.1-3) (SO.sub.4-6)GlcNAc-) by using

transformants which harbors the DNA.

Summary of Invention Paragraph - BSTX (8):

[0005] An object of the present invention is to provide a polypeptide of N-acetylglucosamine-6-O**-sulfotransferase** and a DNA encoding the polypeptide.

Summary of Invention Paragraph - BSTX (9):

[0006] The present inventors have succeeded in cloning a DNA encoding a polypeptide of N-acetylglucosamine-6-O**-sulfotransferase,** having an activity to specifically transfer a sulfate group to a hydroxyl group at 6 position of an N-acetylglucosamine residue located at a non-reducing end of GlcNAc.beta.1-3Gal.beta.1-4GlcNAc, wherein GlcNAc represents an N-acetylglucosamine-residue, Gal represents a galactose residue, .beta.1-3 represents a .beta.1-3 glycosidic linkage, and .beta.1-4 represents a .beta.1-4 glycosidic linkage. The inventors have also confirmed that the polypeptide of N-acetylglucosamine-6-O**-sulfotransferas- e** was expressed by the DNA and identified the polypeptide, thereby completing the present invention.

Detail Description Paragraph - DETX (67):

[0112] Oligonucleotide primers (a sense primer and an antisense primer) are prepared based on a mouse expressed sequence tag (EST) sequence (Genbank accession number, AA103962) having homology to the catalytic site of mouse chondroitin 6**-sulfotransferase**. Specific examples as oligonucleotide primers are

Detail Description Paragraph - DETX (141):

[0180] The polypeptide of the present invention is a polypeptide of N-acetylglucosamine-6-O**-sulfotransferase** having activity to specifically transfer a sulfate group to a hydroxyl group at 6 position of an N-acetylglucosamine residue located at a non-reducing end of a sugar chain. Therefore, it is useful for synthesis of functional sugar chains such as GlyCAM-1 (expected to be used as an anti-inflammatory agent or the like). The DNA of the present invention can be used for synthesis of the polypeptide of the present invention in large scale and artificial expression of functional sugar chains such as GlyCAM-1 in the living organism (cells).

Detail Description Paragraph - DETX (153):

[0191] A mouse expressed sequence tag (EST) sequence (Genbank accession number AA103962) with similarity to the catalytic portion of mouse chondroltin 6**-sulfotransferase** was amplified by the RT-PCR method using mouse day-13 embryo total RNA as a template. The sense primer, GTCGTCGGACTGGTGGACGA (SEQ ID NO:5) and the antisense primer, CCCAGAGCGTGGTAGTCTGC (SEQ ID NO:6), were used for PCR amplification, which was carried out at 94.degree. C. for 3 min, with 35

cycles of 94.degree. C. for 0.5 min, 60.degree. C. for 1 min and 72.degree. C. for 1 min. The PCR product (368 bp) was .sup.32P-labeled with a Megaprime.TM. DNA labeled kit (Amersham Co.) and was used to screen the .lambda.gt 11 mouse day-7 embryo cDNA library.

Detail Description Paragraph - DETX (164):

[0202] (5) Assay of **Sulfotransferase** Activity to Various High Molecular Weight Substrates

Detail Description Paragraph - DETX (165):

[0203] **Sulfotransferase** activities were assayed using various glycosaminoglycans as substrates (acceptors) as described (J. Biol. Chem., 272, 32321-32328 (1997)). When mucins were used as acceptors, reaction mixture containing 2.5 .mu.mol of imidazole-HCl, pH 6.8, 0.25 .mu.mol of CaCl.sub.2, 0.1 .mu.mol of dithiothreitol, 0.1 mmol of NaF, 0.1 .mu.mol of AMP, 2.0 .mu.g of mucins, 50 pmol of .sup.35S-PAPS (about 5.0.times.10.sup.5 cpm), and 5 .mu.l of the extracts in a final volume of 50 .mu.l was incubated at 37.degree. C. for 1 h.

Detail Description Paragraph - DETX (167):

[0205] (6) Assay of **Sulfotransferase** Activity to Oligosaccharides

Detail Description Paragraph - DETX (168):

[0206] The reaction mixture contained 2.5 .mu.mol of imidazole-HCl, pH 6.8, 0.5 .mu.mol of MnCl.sub.2, 0.1 .mu.mol of AMP, 1.0 .mu.mol of NaF, 25 mmol of oligosaccharides, 50 pmol of .sup.35S-PAPS (about 5.times.10 cpm), and 5 .mu.l of the extracts in a final volume of 50 .mu.l. The reaction mixture was incubated at 30.degree. C. for 5 h and the reaction was stopped by immersing the reaction tubes in a boiling water bath for 1 min. .sup.35S-Labeled oligosaccharides were separated from .sup.35SO.sub.4 and .sup.35S-PAPS by Superdex 30 gel chromatography, and the radioactivity was determined. When GlcNAc.beta.1-3Gal.beta.1-4GlcNAc was used as an acceptor, **sulfotransferase** reaction proceeded linearly up to 5 h under the assay conditions.

Detail Description Paragraph - DETX (185):

[0223] Specimens from C57 BL/6J mice were subjected to hematoxylin-eosin staining or in situ hybridization. As the probe for the polypeptide of the present invention, a 0.6 kbp **Pst** I fragment of the cDNA (nucleotide numbers 962 to 1561 in SEQ ID NO:1) was subcloned into pBluescript II SK-. Sense and antisense cRNA probes were prepared by in vitro transcription with a DIG RNA labeling kit (Boehringer Mannheim Co., Germany).

Detail Description Paragraph - DETX (190):

[0228] Mouse chondroitin 6-**sulfotransferase** had cloned previously. By searching in the EST database, we found a small sequence with similarity to the catalystic portion of mouse chondroitin 6-**sulfotransferase** (Genbank accession number AA103962). We obtained the corresponding cDNA fragment by RT-PCR (nucleotide numbers 1139 to 1506 in SEQ ID NO:1). Approximately 8.times.10.sup.5 plaques of a mouse day-7 embryo cDNA library were screened using the cDNA fragment as a probe, and six independent clones were obtained. The nucleotide sequence of the largest cDNA insert (2.2 kb) was determined (SEQ ID NO:1). The determined 2150-bp cDNA had a single open reading frame consisting of 483 amino acids, with a molecular mass of 52829 Da and four potential N-linked glycosylation sites (SEQ ID NO:1). The sequence around the first ATG codon fitted Kozak's rule (Cell, 44, 283-292 (1986)), and the upstream region contained an in-frame stop codon. Hydropathy plot analysis indicated the presence of one prominent hydrophobic segment 20 residues in length in the amino-terminal region (Ala.sup.8-Leu.sup.27) predicting that the polypeptide of the present invention is type II transmembrane protein (FIG. 1). The polypeptide of the present invention showed 25% and 27% homology with mouse chondoroitin 6-**sulfotransferase** and human keratan sulfate Gal-6-**sulfotransferase,** respectively. However, no significant homology in amino acid sequence was observed between the protein and other known **sulfotransferases** (J. Biol. Chem., 267, 15744-15750 (1992), J. Biol. Chem., 272, 13980-13985 (1997), J. Biol. Chem., 272, 28008-28019 (1997), J. Biol. Chem., 272, 29942-29946 (1997), J. Biol. Chem., 272, 4864-4868 (1997)).

Detail Description Paragraph - DETX (196):

[0234] The DNA of the present invention, from which the bulk of the 5'- and 3'-non-coding regions was removed (nucleotide numbers 467 to 1921 in SEQ ID NO:1) was inserted into a mammalian expression vector pcDNA3 and overexpressed in COS-7 cells. Extracts of the transfected cells were assayed for **sulfotransferase** activity using .sup.35S-labeled PAPS as the sulfate group donor and various glycoconjutages as sulfate group acceptors: chondroitin, chondroitin 4-sulfate, chondroitin 6-sulfate, dermantan sulfate, keratan sulfate, desulfated keratan sulfate, CDSNS-heparin, mucin from porcine stomach and mucin from bovine submaxillary gland did not serve as acceptors. We examined GlcNAc.beta.1-3Gal.beta.1-4GlcNAc as a sulfate group acceptor. Superdex 30 chromatography of the reaction mixture indeed revealed a radioactive peak in cells transfected with a vector (pcDNA3-GlcNAc6ST) containig the cDNA of the correct orientation (sense DNA) slighly larger than the acceptor, indicating that the acceptor was sulfated (FIG. 2A). The extract from untransfected cells or those transfected with a vector (pcDNA3-GlcNAc6STA) containing the cDNA of the reverse orientation (the antisense cDNA) showed much less **sulfotransferase** activity (Table 1). The acitivity was calculated from the radioactivity contained in Fraction Number 85 to 89 in Superdex 30 chromatography (FIG. 2A). Values are shown that values obtained in the absence of the acceptor were subtracted from the value obtained in the presence of the acceptor. Values of averages .+-.S.D. of triplicate culture are shown.

Detail Description Paragraph - DETX (198):

[0236] COS-7 cells were transfected with the DNA of the present invention from which the bulk of 5'- and 3'-non-coding region was removed (nucleotide numbers 387 to 1844 in SEQ ID NO:3) in the same manner as described above and the extracts of resulting transfectants were examined for its **sulfotransferase** activity (using GlcNAc.beta.1-3Gal.beta.1-4GlcNA- c.beta.1-3Gal.beta.1-4GlcNAc as a sulfate group acceptor) in the same manner as described above. As a result, the cells transfected with the vector (pcDNA3-hGlcNAc6ST) containing the cDNA with the correct orientation (sense cDNA) exhibited fivefold or more as high **sulfotransferase** activity as untransfected cells or the cells transfected with the vector (pcDNA3-hGlcNAc6STA) containing the cDNA with the reverse orientation (antisense cDNA)(Table 2). The activity was calculated from radioactivity of fractions obtained by Superdex 30 chromatography. Values were shown that values obtained in the absence of the acceptor were subtracted from the value obtained in the presence of the acceptor. Values of averages .+-.S.D. of triplicate culture are shown.

Detail Description Table CWU - DETL (1):

1 TABLE 1 **Sulfotransferase** activity Vector pmole/hour/mg protein None (untransfected) 1.1 .+-. 0.7 Sense cDNA 10.6 .+-. 2.4 Antisense cDNA 1.8 .+-. 0.9

Detail Description Table CWU - DETL (2):

2 TABLE 2 **Sulfotransferase** activity Vector pmole/hour/mg protein None (untransfected) 1.33 .+-. 0.62 Sense cDNA 6.67 .+-. 0.51 Antisense cDNA 1.29 .+-. 0.23

PGPUB-DOCUMENT-NUMBER: 20030008354

PGPUB-FILING-TYPE: new

DOCUMENT-IDENTIFIER: US 20030008354 A1

TITLE: Isolated human drug-metabolizing proteins, nucleic acid molecules encoding human drug-metabolizing proteins, and uses thereof

PUBLICATION-DATE: January 9, 2003

INVENTOR-INFORMATION:

NAME	CITY	STATE	COUNTRY	RULE-47
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APPL-NO: 10/ 199334

DATE FILED: July 22, 2002

RELATED-US-APPL-DATA:

child 10199334 A1 20020722

parent division-of 09609816 20000703 US GRANTED

parent-patent 6436684 US

non-provisional-of-provisional 60212725 20000620 US

non-provisional-of-provisional 60192408 20000327 US

US-CL-CURRENT: 435/69.1, 435/183 , 435/320.1 , 435/325 , 530/350 , 536/23.2

ABSTRACT:

The present invention provides amino acid sequences of peptides that are encoded by genes within the human genome, the proteins of the present invention. The present invention specifically provides isolated peptide and nucleic acid molecules, methods of identifying orthologs and paralogs of the proteins of the present invention, and methods of identifying modulators of the proteins of the present invention.

---------- KWIC ---------

Summary of Invention Paragraph - BSTX (34):

[0032] In general, sulfation is a deactivating, detoxication pathway, but for some chemicals the sulfate conjugates are much more reactive than the parent compound (Coughtrie M W, et al., 1998. Biology and function of the reversible sulfation pathway catalysed by human sulfotransferases and sulfatases. Chem Biol Interact. 20;109(1-3):3-27). For example N-hydroxyarylamine **sulfotransferase (HAST-I) detoxifies phenols** but activates N-hydroxylarylamines into a mutagenic form, N-hydroxy-2-acetylaminofluorene (Nagata, K., et al., 1993. Isolation and expression of a cDNA encoding a male-specific rat sulfotransferase that catalyzes activation of N-hydroxy-2-acetylaminofluorene. J. Biol. Chem. 268 (33), 24720-24725 and Gong D W, et al., 1991. Purification of hepatic N-hydroxyarylamine sulfotransferases and their regulation by growth hormone and thyroid hormone in rats. J Biochem (Tokyo) 1991 August; 110(2):226-31.).

PGPUB-DOCUMENT-NUMBER: 20030003472

PGPUB-FILING-TYPE: new

DOCUMENT-IDENTIFIER: US 20030003472 A1

TITLE: Mismatch repair detection

PUBLICATION-DATE: January 2, 2003

INVENTOR-INFORMATION:

NAME	CITY	STATE	COUNTRY	RULE-47
Cox, David R.	Belmont	CA	US	
Faham, Malek	Daly City	CA	US	
Baharloo, Siamak	San Francisco	CA	US	

APPL-NO: 10/ 081771

DATE FILED: February 20, 2002

RELATED-US-APPL-DATA:

child 10081771 A1 20020220

parent continuation-in-part-of 09271055 19990317 US GRANTED

parent-patent 6406847 US

child 09271055 19990317 US

parent continuation-in-part-of 08713751 19960913 US ABANDONED

non-provisional-of-provisional 60004664 19951002 US

US-CL-CURRENT: 435/6, 435/471 , 435/484 , 435/488

ABSTRACT:

Mismatch Repair Detection (MRD), a novel method for DNA-variation detection, utilizes bacteria to detect mismatches by a change in expression of a marker gene. DNA fragments to be screened for variation are cloned into two MRD plasmids, and bacteria are transformed with heteroduplexes of these constructs. Resulting colonies express the marker gene in the absence of a mismatch, and-lack expression in the presence of a mismatch. MRD is capable of detecting a single mismatch within 10 kb of DNA. In addition, MRD can analyze many fragments simultaneously, offering a powerful method for high-throughput genotyping and mutation detection.

CROSS-REFERENCE TO RELATED APPLICATIONS

[0001] This application is a continuation-in-part of U.S. patent application

Ser. No. 09/271,055, filed Mar. 17, 1999, now allowed, which is a continuation-in-part of U.S. patent application Ser. No. 08/713,751, filed Sep. 13, 1996, which claims priority to U.S. Provisional Patent Application No. 60/004,664, filed Oct. 2, 1995, the disclosures of which are incorporated herein by reference in their entireties.

---------- KWIC ---------

Detail Description Paragraph - DETX (292):

[0352] Other useful loci that can provide standards and as to which loci sequence variation can be queried include EPHX1 (epoxide hydrolase 1, microsomal xenobiotic), EPHX2 (epoxide hydrolase 2), LTA4H (leukotriene A4 hydrolase), TRAG3 (Taxol.RTM. resistance associated gene 3, which is overexpressed in most melanoma cells and confers resistance to paclitaxel, Taxol.RTM.), GUSB (beta-glucuronidase), TMPT (thiopurine methyltransferase), BCRP, (breast cancer resistance protein, an ATP transporter), dihydropyrihidine dehydrogenase, HERG (involved in drug transport through potassium ion channels), hKCNE2 (involved in drug transport through potassium ion channels), UDP glucuronosyl transferase (UGT) (a hepatic metabolizing enzyme, a detoxifying enzyme for most carcinogens after different cytochrome P450 (CYP) isoforms), **sulfotransferase,** sulfatase, and glutathione S-transferase (GST) -alpha, -mu, -pi (which detoxify therapeutic drugs, not least several anti-cancer drugs), ACE (peptidyl-dipeptidase A), and KCHN2 (potassium voltage-gated channel, subfamily H (eag-related), member 2), location 7q35-q36).

Detail Description Paragraph - DETX (370):

[0422] The Cre gene was PCR amplified using a construct carrying Cre as a template. The PCR left 13 bp 5' of the start ATG of the protein. The PCR product was cloned in **Pst**/BamHI digested pBSK (Stratagene). The Cre gene was then used to replace the LacZ.alpha. gene in pMF200. To this end the Cre fragment was released by an Eco RI/Xba I double digestion and the ends were filled using the Klenow fragment. PMF200 was partially digested with Bgl I and treated with T4 DNA polymerase to chew the 3' overhang followed by Bam HI digest (the Bam HI site is present in the leader sequence of LacZ.alpha. of pMF100) and end filling by Klenow fragment. A blunt end ligation produced pMRD100, which replaced the LacZ.alpha. gene on the Bgl I-Bam HI fragment of pMF100 by the Cre gene. In this construct the plac promoter drives the expression of Cre.

PGPUB-DOCUMENT-NUMBER: 20020182681

PGPUB-FILING-TYPE: new

DOCUMENT-IDENTIFIER: US 20020182681 A1

TITLE: Isolated human drug-metabolizing proteins, nucleic acid molecules encoding human drug-metabolizing proteins, and uses thereof

PUBLICATION-DATE: December 5, 2002

INVENTOR-INFORMATION:

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APPL-NO: 10/ 199330

DATE FILED: July 22, 2002

RELATED-US-APPL-DATA:

child 10199330 A1 20020722

parent division-of 09609816 20000703 US GRANTED

parent-patent 6436684 US

non-provisional-of-provisional 60212725 20000620 US

non-provisional-of-provisional 60192408 20000327 US

US-CL-CURRENT: 435/69.1, 435/183 , 435/320.1 , 435/325 , 530/350 , 536/23.2

ABSTRACT:

The present invention provides amino acid sequences of peptides that are encoded by genes within the human genome, the proteins of the present invention. The present invention specifically provides isolated peptide and nucleic acid molecules, methods of identifying orthologs and paralogs of the proteins of the present invention, and methods of identifying modulators of the proteins of the present invention.

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Summary of Invention Paragraph - BSTX (34):

[0032] In general, sulfation is a deactivating, detoxication pathway, but for some chemicals the sulfate conjugates are much more reactive than the parent compound (Coughtrie M W, et al., 1998. Biology and function of the reversible sulfation pathway catalysed by human sulfotransferases and sulfatases. Chem Biol Interact. 20;109(1-3):3-27). For example N-hydroxyarylamine **sulfotransferase (HAST-I) detoxifies phenols** but activates N-hydroxylarylamines into a mutagenic form, N-hydroxy-2-acetylaminofluorene (Nagata, K., et al., 1993. Isolation and expression of a cDNA encoding a male-specific rat sulfotransferase that catalyzes activation of N-hydroxy-2-acetylaminofluorene. J. Biol. Chem. 268 (33), 24720-24725 and Gong D W, et al., 1991. Purification of hepatic N-hydroxyarylamine sulfotransferases and their regulation by growth hormone and thyroid hormone in rats. J Biochem (Tokyo) 1991 Aug;110(2):226-31.).

PGPUB-DOCUMENT-NUMBER: 20020168771

PGPUB-FILING-TYPE: new

DOCUMENT-IDENTIFIER: US 20020168771 A1

TITLE: Vectors having replication, immunogenicity and/or pathogenicity under stress promoter regulation and use thereof

PUBLICATION-DATE: November 14, 2002

INVENTOR-INFORMATION:

NAME	CITY	STATE	COUNTRY	RULE-47
Gamerman, Gary Eric	Vienna	VA	US	

APPL-NO: 09/ 850270

DATE FILED: May 8, 2001

US-CL-CURRENT: 435/456, 435/235.1 , 435/320.1

ABSTRACT:

The present invention relates to modified vectors, e.g. plasmids, viruses or microbia sucsh as yeast or bacteria, wherein the replication, immunogenicity and/or pathogenicity is placed under the control of at least one stress gene regulating element. In preferred embodiments, these modified vectors are used for gene therapy, in vaccines, or for functional genomic screening.

---------- KWIC ---------

Summary of Invention Paragraph - BSTX (145):

[0141] 11-beta hydroxysteroid dehydrogenase type II, 12-lipoxygenase, 17-beta hydroxysteroid dehydrogenase, 60S ribosomal protein L6,6-Omethylguanine -DNA methyltransferase, Activating transcription factor 2, Activating transcription factor 3, Activating transcription factor 4, Activin beta E, Activin receptor type 11, Acyl-CoA dehydrogenase, Acyl CoA Carrier Protein, Adenine nucleotide translocator 1, Alanine aminotransferase, Alcohol dehydrogenase 1, Alcohol dehydrogenase 2, Alcohol dehydrogenase 3, Alcohol dehydrogenase 4, Alcohol dehydrogenase 5, Aldehyde dehydrogenase 1, Aldehyde dehydrogenase 2, Aldehyde dehydrogenase 3, Alpha 1-antitrypsin, Alpha-1 acid glycoprotein, Alpha-1 antichymotrypsin, Alpha-catenin, Alphatubulin, Apolipoprotein A1, Apolipoprotein A11, Apolipoprotein CIil, Apolipoprotein E, Aryl hydrocarbon receptor, Aspartate aminotransferase, mitochondrial, Ataxia telangeictasia, ATP-dependent helicase 11 (70 kDa), ATP-dependent helicase 11 (Ku80), BAG-1, BAK, Bax (alpha), Bcl-2, Bcl-xL, Beta-actin, Bilirubin UDP-glucuronosyl-transferase isozyme 1, Bilirubin UDP-glucuronosyl-transferase

isozyme 2, Biliverdin reductase, Branched chain acylCoA oxidase, BRCA1, BR-cadherin, C4bbinding protein, c-abl, Calcineurin B, Calnexin, Calprotectin, Calreticulin, canalicular multispecific organic anion transporter, Carbonic Anhydrase 111, Carnitine palmitoyl-CoA transferase, Caspase 1, Caspase 2 (Nedd2), Caspase 3 (CPP32beta), Caspase 5 (ICE relIII), Caspase 6 (Mch2-alpha), Caspase 7 (Mch3alpha), Caspase 8 (FLICE), Catalase, CatecholOmethyltransferase, CCAAT/enhancer-binding protein alpha, CCAAT/enhancer-binding protein epsilon, Cell division cycle protein 2, Cell division cycle protein 20, Cell division cycle protein 25, Cellular retinoic acid binding protein 1, Cellular retinoic acid binding protein 2, cerb; c-fos, Checkpoint kinase-1, Cholesterol esterase, c-H-ras, cjun, Clusterin, c-myc, Complement component C3, Connexin 30, Connexin32, Connexin-40, Corticosteroid binding globulin, Corticotropin releasing factor, C-reactive protein, Creatine kinase b, Cyclin D1, Cyclin dependent kinase 1, Cyclin dependent kinase 4, Cyclin dependent kinase inhibitor 1A, Cyclin E, Cyclin G, Cyclin-dependent kinase 4 inhibitor (P116), Cyclindependent kinase 4 inhibitor B (P16), Cyclin-dependent kinase inhibitor P27Kip1, Cyclooxygenase 2, Cystic fibrosis transmembrane conductance regulator, Cytochrome P450 11A1, Cytochrome P450 17A, Cytochrome P450 1A1, Cytochrome P450 1A2, Cytochrome P450 1 B1, Cytochrome P450 2A1, Cytochrome P450 2A3, Cytochrome P450 2A6, Cytochrome P450 2131, Cytochrome P450 21310, Cytochrome P450 2132, Cytochrome P450 2C11, Cytochrome P450 2C12, Cytochrome P450 2C19, Cytochrome P450 2C9, Cytochrome P450 2D6, Cytochrome P450 2E1, Cytochrome P450 2F2, Cytochrome P450 3A1, Cytochrome P450 3A4, Cytochrome P450 4A, Cytochrome P450 4A1, Damage specific DNA binding protein p48 subunit, Defender against cell death-1, Deleted in colorectal cancer, Deltalike protein, Dihydrofolate reductase, Disulfide isomerase related protein (ERp72), DNA binding protein inhibitor ID2, DNA dependent helicase, DNA dependent protein kinase, DNA ligase 1, DNA ligase IV, DNA mismatch repair protein (MLH1), DNA mismatch repair protein (PMS2), DNA mismatch repair/binding protein (MSH3), DNA polymerase alpha, DNA polymerase beta, DNA polymerase beta, DNA repair and recombination homologue (RAD 52), DNA repair helicase II ERCC-3, DNA repair protein (RAD 50), DNA repair protein (XRCC1), DNA repair protein XP-D, DNA replication factor C (36 kDa), DNA topoisomerase 1, DNA topoisomerase 11, Dopamine beta-hydroxylase, DRA, Dynein light chain 1, E2F, Early growth regulated protein 1, E-Cadherin, ECE-1 (endothelin converting enzyme), Endothelin-1, Enolase alpha, Enoyl CoA hydratase, Eotaxin, Epidermal growth factor, Epoxide hydrolase, ERA-B, ERCC 1 (excision repair protein), ERCC 3 (DNA repair helicase 11), ERCC 5 (excision repair protein), ERCC 6 (excision repair protein), ERK1, Erythropoietin, Erythropoietin receptor, ESelectin, Estrogen receptor, Farnesol receptor, Fas antigen, Fas associated death domain (FADD), Fas ligand, Fas/Apo1 receptor, Fatty acid synthase, Fatty acyl-CoA oxidase, Fatty acyl-CoA synthase, FEN-1 (endonuclease), Fibrinogen gamma chain, Fibronectin receptor, FIC1, Filagrin, Flavin containing monooxygenase 1, Flavin containing monooxygenase 3, FosB, Fra-1, Fucosyl transferase (alpha-1,2fucosyltransferase), Gadd153, Gadd45, Gamma-glutamyl hydrolase precursor, Gamma-glutamyl transpeptidase, GCLR, GCLS, Glucocorticoid receptor, Glucose-6-phosphate dehydrogenase, Glucose-regulated protein 170, Glucose-regulated protein 58, Glucose-regulated protein 78, Glucoseregulated protein 94, Glutamicoxaloacetic transaminase, Glutaminc-pyruvic transaminase, Glutathione peroxidase, Glutathione reductase, Glutathione S-transferase alpha subunit, Glutathione S-transferase 4a, Glutathione synthetase, Glyceraldehyde 3-phosphate dehydrogenase, GOS24 (zinc finger transcriptional regulator), Granulocyte-macrophage colony-stimulating factor, Growth-arrested-specifi- c protein 1, Growth-arrested-specific protein

3, GT mismatch binding protein, H-cadherin, Heat shock protein 12, Heat shock protein 47, Heat shock protein 70, Heat shock protein 70.1, Heat shock protein 90, Helicase-like transcription factor, Heme binding protein 23, Heme oxygenase-1, Hepatic lipase, Hepatocyte growth factor, Hepatocyte growth factor activator, Hepatocyte growth factor receptor, Hepatocyte nuclear factor 4, Histone 2A, Histone 28, HMG CoA reductase, Hydroxyacyl CoA dehydrogenase, Hydroxysteroid sulfotransferase a, Hypoxanthine-guanine phosphoribosyltransferase, ICE-rel 11 (Caspase 4), ICH-2 cysteine protease=CASPASE 4, IkB-a, Insulin-like growth factor binding protein 1, Insulin-like growth factor binding protein 2, Insulin-like growth factor binding protien 3, Insulin-like growth factor I, Insulin-like growth factor 11, Integrin alpha, Integrin alpha L, Integrin betas, Integrin beta2, Intercellular adhesion molecule-1, Intercellular adhesion molecule-2, Intercellular adhesion molecule-3, Interferon gamma, Interferon inducible protein 10, Interferon inducible protein 15, Interleukin-1 alpha, Interleukin-12, Interleukin-2, Interleukin-4, Interleukin-5, Interleukin-6, Involucrin, JNK1 stress activated protein kinase, K-cadherin, Ki67, Lactate Dehydrogenase 8, Lactoferrin, Lipopolysaccharide binding protein, Lipoprotein lipase precursor, Liver fatty acid binding protein, L-myc, Low density lipoprotein receptor, Luteinizing hormone, Lysyl oxidase, Macrophage inflammatory protein-1 alpha, Macrophage inflammatory protein-1 beta, Macrophage inflammatory protein-2 alpha, Macrophage inflammatory protein-2 beta, Macrophage inflammatory protein-3 alpha, Macrophage inflammatory protein-3 beta, Malic enzyme, MAP kinase kinase, Matrix metal loproteinase1, Matrix metal loproteinase-2, MDM-2, MET proto-oncogene, Metallothionein 1, Metallothionein 2, Metallothionein 3, Metallothionein IA, Metallothionein IG, Metalregulatory transcription factor-1, Mitogen activated protein kinase (P38), Mitogen inducible gene (mig-2), MOAT-B (MRP/organic anion transporter), Monoamine oxidase A, Monoamine oxidase B, Multidrug resistance-associated protein, Multidrug resistant protein-1, Multidrug resistant protein-2, Multidrug resistant protein-3 =cMOAT2, MUTL homologue (MLH1), MutS Homologue (MSH2), Myeloid cell differentiation protein-1, Na/taurocholate cotransporting polypeptide, NADPH cytochrome P450 -oxidoreductase, NADPH cytochrome P450 reductase, NADPH quinone oxidoreductase-1 (DTDiaphorase), Natural killer cell-enhancing factor B, N-cadherin, NF-kappaB (p65), Nitric oxide synthase-1, inducible, Nucleoside diphosphate kinase beta isoform, 0-6-alkylguanine-DNAalkyltran- sferase, OBcadherin 1, OB-cadherin 2, Octamer binding protein 1, Octamer binding protein 2, Octamer binding protein 3, Oncostatin M, Organic anion transporter 1, Organic anion transporter 3, Organic anion transporter K1, Organic anion transporting polypeptide 1, Organic cation transporter 1, Organic cation transporter 2, Organic cation transporter 3, Organic cation transporter N1, Organic cation transporter N2, Ornithine decarboxylase, Osteopontin, Oxygen regulated protein 150, p53, PAPS synthetase, P-cadherin, PEGS (progression elevated gene 3), Peroxisomal 3-ketoacyl-CoA thiolase 1, Peroxisomal 3-ketoacylCoA thiolase 2, Peroxisomal acyl-CoA oxidase, Peroxisomal fatty acyl-CoA oxidase, Peroxisome assembly factor 1, Peroxisome assembly factor 2, Peroxisome biogenesis disorder protein-1, Peroxisome biogenesis disorder protein-11, Peroxisome biogenesis disorder protein-4, Peroxisome hydratase, Peroxisome proliferator activated receptor alpha, Peroxisome proliferator activated receptor gamma, **Phenol sulfotransferase,** Phosphoenolpyruvate carboxykinase, Phosphoglyceride kinase, Phospholipase A2, Plasminogen activator inhibitor 2, Platelet derived growth factor B, Platelet/endothelial cell adhesion molecule-1, Poly (ADP ribose) polymerase, Proliferating cell nuclear

antigen gene, Prostaglandin H synthase, Protein kinase C betal, Protein-tyrosine phosphatase, Putative protein tyrosine phosphatase, RAID, RAID 51 homologue, RANTES, Ref 1, Replication factor C, 40-kDa subunit (Al), Replication protein A (70 kDa subunit), Retinoblastoma, Retinoblastoma related protein (P 107), Retinoid X receptor alpha, Retinoid X receptor beta, Retinoid X receptor gamma, Ribonucleotide reductase M1 subunit, Ribosomal protein L13A, Ribosomal protein S9, RNA-dependent helicase, ROAT1 (renal organic anion transporter), Serum amyloid A1, Serum amyloid A2alpha, Sister of p-glycoprotein, Sodium/bile acid cotransporter, Sonic hedgehog gene, SQM1, Superoxide Dismutase Cu/Zn, Superoxide dismutase Mn, T-cell cyclophilin, Tenascin, Thiopurine methyltransferase, Thioredoxin, Thrombospondin 2, Thymidine kinase, Thymidylate synthase, Thymosin beta-10, Tissue inhibitor of metalloproteinases-1, Tissue transglutaminase, Transcription factor IID, Transferrin, Transforming growth factor-beta 3, Tumor necrosis factor associated factor 2 (TRAF2), Tumor necrosis factor receptor 1, Tumor necrosis factor receptor 2, Tumor necrosis factor receptor-1 associated protein (TRADD), Tumor necrosis factor-alpha, Tumor necrosis factorbeta, Type 1 interstitial collagenase, Tyrosine aminotransferase, Tyrosine protein kinase receptor (UFO), Ubiquitin, Ubiquitin conjugating enzyme (Rad 6 homologue), Ubiquitin-homology domain protein PIC1, UDPglucuronosyltransferase 1, UDP-glucuronosyltransferase 1A6, UDPglucuronosyltransferase 2, UDP-glucuronosyltransferase 28, Uncoupling protein 1, Uncoupling protein 2, Uncoupling protein 3, Urate oxidase, UV excision repair protein RAD 23 (XP-C), Vascular cell adhesion molecule 1 (VCAM-1), Vascular endothelial growth factor, Vascular endothelial growth factor D, Very long-chain acyl-CoA dehydrogenase, Vimentin, Vitellogenin, Waf1, XRCC1 (DNA repair protein).

PGPUB-DOCUMENT-NUMBER: 20020164748

PGPUB-FILING-TYPE: new

DOCUMENT-IDENTIFIER: US 20020164748 A1

TITLE: Glycosyl sulfotransferase-3

PUBLICATION-DATE: November 7, 2002

INVENTOR-INFORMATION:

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Hemmerich, Stefan	Berkeley	CA	US	

APPL-NO: 10/ 007262

DATE FILED: November 8, 2001

RELATED-US-APPL-DATA:

child 10007262 A1 20011108

parent division-of 09190911 19981112 US GRANTED

parent-patent 6365365 US

child 09190911 19981112 US

parent continuation-in-part-of 09045284 19980320 US GRANTED

parent-patent 6265192 US

US-CL-CURRENT: 435/193, 435/320.1 , 435/325 , 435/69.1 , 536/23.2

ABSTRACT:

A novel human glycosylsulfotransferase expressed in high endothelial cells (GST-3) and polypeptides related thereto, as well as nucleic acid compositions encoding the same, are provided. The subject polypeptides and nucleic acid compositions find use in a variety of applications, including research, diagnostic, and therapeutic agent screening applications. Also provided are methods of inhibiting selectin mediated binding events and methods of treating disease conditions associated therewith, particularly by administering an inhibitor of at least one of GST-3 or KSGal6ST, or homologues thereof.

CROSS-REFERENCE TO RELATED APPLICATIONS

[0001] This application is a continuation-in-part of application Ser. No.

09/045,284 filed on Mar. 20, 1998, the disclosure of which is herein incorporated by reference.

---------- KWIC ---------

Summary of Invention Paragraph - BSTX (5):

[0005] Sulfotransferases are enzymes that catalyze the transfer of a sulfate from a donor compound to an acceptor compound, usually placing the sulfate moiety at a specific location on the acceptor compound. There are a variety of different sulfotransferases which vary in activity, i.e. with respect to the donor and/or acceptor compounds with which they work. Known sulfotransferases include those acting on carbohydrate: heparin/heparan sulfate N-sulfotransferase COST); chondroitin 6/keratan 6 sulfate sulfotransferase (C6ST/KSST); galactosylceramide 3'-sulfotransferase; heparan sulfate 2-sulfotransferase (Iduronic acid); HNK-1 sulfotransferase (3-glucuronic acid); heparan sulfate D-glucosamino 3-O-sulfotransferase (3-OST);etc., as well as those acting on **phenols, steroids and xenobiotics: aryl sulfotransferase** I & II, hydroxy-steroid sulfotransferases I, II & III, dehydroepiandrosterone (DHEA); etc. Sulfotransferases play a central role in a variety of different biochemical mechanisms, as the presence of a sulfate moiety on a particular ligand is often required for a particular activity, e.g. binding.

US-PAT-NO: 6534313

DOCUMENT-IDENTIFIER: US 6534313 B1

TITLE: Genetically modified plants having modulated brassinosteroid signaling

DATE-ISSUED: March 18, 2003

INVENTOR-INFORMATION:

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Neff; Michael M.	St. Louis	MO	N/A	N/A
Chory; Joanne	Del Mar	CA	N/A	N/A

APPL-NO: 09/ 527073

DATE FILED: March 16, 2000

PARENT-CASE:

RELATED APPLICATIONS

This application claims priority under Section 119(e)(1) to U.S. Provisional Patent Application Serial No. 60/172,832, filed Dec. 20, 1999; U.S. Provisional Patent Application Serial No. 60/170,931, filed Dec. 14, 1999; and U.S. Provisional Patent Application Serial No. 60/124,570, filed Mar. 16, 1999.

US-CL-CURRENT: 435/419, 435/320.1 , 536/23.6

ABSTRACT:

The present invention provides cytochrome P450s useful in for producing genetically modified plants characterized as having the phenotypic trait of modulated brassinolide synthesis or signaling, for example, resulting in insect resistance, dwarfism and darker-green foliage compared with wild type plants. Such plants can be modified, for example, using "bas1", or functional homologues thereof, a polypeptide encoded by bas1 that modulates brassinolide synthesis and/or signaling in plants. The invention also provides methods for modulating ecdysteroid activity in a plant and for assaying brassinosteroid function in a plant. The latter method can be used to create a gain-of-function allelic series of plants characterized by increasing levels of overexpression of a cytochrome P450 to screen for brassinolide activity in a plant species.

11 Claims, 13 Drawing figures

Exemplary Claim Number: 1

Number of Drawing Sheets: 7

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Detailed Description Text - DETX (65):

In another embodiment, the invention provides a method for producing a genetically modified plant characterized as having increased disease or insect as compared to a plant which has not been genetically modified (e.g., a wild-type plant). The term "disease or insect" or "pathogen" or "insect" resistance refers to the ability to maintain a desirable phenotype upon exposure to infection, relative to a nontransgenic plant. The level of resistance can be determined by comparing the physical characteristics of the invention plant to nontransgenic plants that either have or have not been exposed to infection or insect infestation. Exemplary physical characteristics to observe include an increase in population of plants that have the ability to survive pathogen challenge, delayed lesion development, reduced lesion size, and the like. The term "disease" refers to a pathogen challenge caused any agent known to cause symptoms of infection in plants, including, but not limited to bacteria, nematodes, viruses, mycoplasmas, and fungi. In a preferred embodiment, the pathogen is a bacterial pathogen, including, but not limited to, Pseudomonas. Exemplary organisms include Pseudomonas synringe pv. tomato (**Pst**) and Pseudomonas syringe pv. maculicola (Psm). The term "increased resistance to pathogens" or "increased resistance to disease" refers to a level of resistance that an invention transgenic plant has to plant pathogens above a defined reference level such as the level of resistance displayed by nontransgenic plants of the same species. Thus, the increased resistance is measured relative to previously existing plants of the same species. In one embodiment, the resistance is substantially increased above the defined reference level greater than or equal to a 20% increase, preferably greater than or equal to a 50% increase, more preferably greater than or equal to a 75% increase, with the most preferred being a 95% increase and above. The phase "nontransgenic plant of the same species" means a plant of the same species that does not contain any heterologous transgenes, or does not contain any transgenes containing a sequence derived from BAS1. The term "heterologous nucleic acid sequence" as used herein refers to a nucleic acid foreign to the recipient plant host or, native to the host if the native nucleic acid is substantially modified from its original form. The levels of pathogen resistance can be determined using methods well known to one of skill in the art. These methods include bacterial resistance assays and fungal infection assays described in U.S. Pat. No. 5,530,187, herein incorporated by reference.

Detailed Description Text - DETX (198):

There is at least one other CYP72 in Arabidopsis (chibi2) that when overexpressed confers a brassinosteroid-minus phenotype similar to bas1-D mutants. Given the genetic redundancy in this brassinolide catabolic process, it is not surprising that bas1-D and chibi2 were both isolated in gain-of-function mutant screens. C26-hydroxylation is probably not the only pathway for inactivating brassinosteroids. Steroid **sulfotransferases** have been

isolated from both Brassica napus and Arabidopsis and shown to inactivate brassinolides through O-sulfonation (M. Rouleau et al., J Biol Chem 274:20925-30, 1999). Though an in vivo role for these **sulfotransferases** has yet to be determined, it is clear that there are multiple mechanisms for the control of brassinosteroids through their catabolism.